Task Order No. 4
Remedial Investigations/
Feasibility Studies
Contract Number
DAAA15-88-D-0009

U S A T H A M A

MILAN ARMY AMMUNITION PLANT

Remedial Investigation Report

VOLUME IV - APPENDICES M - T

FINAL DOCUMENT

December 1991

VOLUME IV

TABLE OF CONTENTS

APPENDIX M

QA/QC DOCUMENTATION

EPA TAL/TCL AND EXPLOSIVES CONSTITUENTS

EPA TCL VOLATILE COMPOUNDS

Chloromethane
Bromomethane
Vinyl Chloride
Chloroethane
Methylene Chloride
Acetone
Carbon Disulfide
1,1-Dichloroethene
1,1-Dichloroethane
1,2-Dichloroethene (total)
Chloroform
1,2-Dichloroethane
2-Butanone
1,1,1-Trichloroethane
Carbon Tetrachloride
Vinyl Acetate
Bromodichloroemethane
1,2-Dichloropropane
cis-1,3-Dichloropropene
Trichloroethene
Dibromochloromethane
1,1,2-Trichloroethane
Benzene
trans-1,3-Dichloropropene
Bromoform
4-Methyl-2-pentanone
2-Hexanone
Tetrachloroethene
Toluene
1,1,2,2-Tetrachloroethane
Chlorobenzene
Ethyl Benzene
Styrene
Xylenes (Total)

EPA TCL SEMIVOLATILE COMPOUNDS

Phenol
bis(2-Chloroethyl)ether
2-Chlorophenol
1,3-Dichlorobenzene
1,4-Dichlorobenzene
Benzyl Alcohol
1,2-Dichlorobenzene
2-Methylphenol
bis(2-Chloroisopropyl)ether
4-Methylphenol
N-Nitrosodipropylamine
Hexachloroethane
Nitrobenzene
Isophorone
2-Nitrophenol
2,4-Dimethylphenol
Benzoic Acid
bis(2-Chloroethoxy)methane
2,4-Dichlorophenol
1,2,4-Trichlorobenzene
aphthalene
-Chloroaniline
Hexachlorobutadiene
4-Chloro-3-methylphenol
2-Methylnaphthalene
Hexachlorocyclopentadiene
2,4,6-Trichlorophenol
2,4,5-Trichlorophenol
2-Chloronaphthalene
2-Nitroaniline
Dimethylphthalate
Acenaphthylene
3-Nitroaniline
Acenaphthene
2,4-Dinitrophenol
4-Nitrophenol
Dibenzofuran
2,4-Dinitrotoluene
Diethylphthalate
4-Chlorophenyl-phenyl ether
Fluorene
4-Nitroaniline
4,6-Dinitro-2-methylphenol
N-Nitrosodiphenylamine
4-Bromophenyl-phenyl ether
Hexachlorobenzene
Pentachlorophenol
Phenanthrene
Anthracene
Di-n-butylphthalate
Fluoranthene
Pyrene
Butylbenzylphthalate
3,3'-Dichlorobenzidine
Benzo[a]anthracene
Chrysene
bis(2-Ethylhexyl)phthalate
Di-n-octylphthalate
Benzo[b]fluoranthene
Benzo[k]fluoranthene
Benzo[a]pyrene
Indeno[1,2,3-cd]pyrene
Dibenz(a,h)anthracene
Benzo[g,h,i]perylene
alpha-BHC
beta-BHC
delta-BHC
gamma-BHC (Lindane)
Heptachlor
Aldrin
Heptachlor Epoxide
Endosulfan I
Dieldrin
4,4'-DDE
Endrin
Endosulfan II
4,4'-DDE
Endosulfan Sulfate
4,4'-DDT
Endrin Ketone
Methoxychlor
alpha-Chlordane
gamma-Chlordane
Toxaphene
Aroclor-1016
Aroclor-1221
Aroclor-1232
Aroclor-1242
Aroclor-1248
Aroclor-1254
Aroclor-1260

EXPLOSIVE ANALYTES

1,3,5-Trinitrobenzene
Cyclotetramethylene Tetranitramine (HMX)
1,3,5-Trinitro-1,3,5-Triazacyclohexane (RDX)
2,4,6-Trinitrophenylmethylnitramine (Tetryl)
Trinitrotoluene (TNT)
Nitrobenzene
2,4-Dinitrotoluene
2,6-Dinitrotoluene
1,3-Dinitrobenzene

TAL INORGANICS

ICP METALS	GFAA METALS	CVAA	COLORIMETRIC
Aluminum	Arsenic	Mercury	Cyanide
Antimony	Lead		
Barium	Selenium		
Beryllium	Silver		
Cadmium	Vanadium		
Calcium			
Chromium			
Copper			
Iron			
Lead			
Magnesium			
Manganese			
Nickel			
Potassium			
Sodium			
Zinc			

USATHAMA CERTIFIED AND UPPER REPORTING LIMITS

USATHAMA REPORTING LIMITS FOR EPA TCL VOLATILES

ANALYTE	USATHAMA ACRONYM	REPORTING LIMIT SOIL[1] (µg/g)	REPORTING LIMIT WATER[2] (µg/L)	UPPER REPORTING LIMIT SOIL (µg/g)	UPPER REPORTING LIMIT WATER (µg/L)
Methylene chloride	CH2CL2	12.0	2.3	200	100
1,1-Dichlororethane	11DCLE	2.3	0.68	200	200
trans-1,2-Dichloroethylene	12DCE	3.0	0.5**	100	200**
1,1-Dichloroethylene	11DCE	3.9	0.5	200	200
Chloroform	CHCL3	0.87	0.5	200	50
1,2-Dichloroethane	12DCLE	1.7	0.5	200	200
1,1,1-Trichloroethane	111TCE	0.5	0.5	200	200
Carbon tetrachloride	CCL4	7.0	0.58	200	200
Trichloroethylene	TRCLE	2.8	0.5	200	200
Benzene	C6H6	1.5	0.5	200	200
1,1,2-Trichloroethane	112TCE	5.4	1.2	200	200
Tetrachloroethylene	TCLEE	0.81	1.6	200	200
Toluene	MEC6H5	0.78	0.5	200	200
Chlorobenzene	CLC6H5	0.86	0.5	200	200
Ethylbenzene	ETC6H5	1.7	0.5	200	200
1,2-Dichloropropane	12DCLP	2.9	0.5	200	200
cis-1,3-Dichloropropylene	C13DCP	3.2	0.58	248	230
Vinyl chloride	C2H3CL	6.2	2.6	200	200
Chloroethane	C2H5CL	12.0	1.9	200	200
Chloromethane	CH3CL	8.8	3.2	100	200
Bromoform	CHBR3	6.9	2.6	200	200
Dibromochloromethane	DBRCLM	3.1	0.67	200	100
trans-1,3-Dichloropropene	T13DCP	2.8	0.70	152	280
1,1,2,2-Tetrachloroethane	TCLEA	2.4	0.51	200	200
Bromodichloromethane	BRDCLM	2.9	0.59	200	200
Bromomethane	CH3BR	5.7	5.8	200	100
Acetone	ACET	17.0	13.0	100	200
Carbon disulfide	CS2	4.4	0.5	100	200
2-Butanone	MEK	70.0	6.4	200	200
Vinyl acetate	CH2AVE	3.2	8.3	100	50
4-Methyl-2-pentanone	MIBK	27.0	3.0	100	200
Styrene	STYR	2.6	0.5	200	200
Xylene	XYLEN	1.5	0.84	200	200

LEGEND:

[1] USATHAMA Method LM19.
[2] USATHAMA Method UM20.
* Detection limit for non-certified compounds.
** The analysis in water detects <u>total</u> 1,2-Dichloroethylene and is not specific to the <u>trans</u> isomer.

USATHAMA REPORTING LIMITS FOR EPA TCL SEMIVOLATILES

ANALYTE	USATHAMA ACRONYM	REPORTING LIMIT		UPPER REPORTING LIMIT	
		SOIL[1] (µg/g)	WATER[2] (µg/L)	SOIL (µg/g)	WATER (µg/L)
Phenol	PHENOL	0.11	9.2	3.3	200
Bis(2-chloroethyl)ether	B2CLEE	0.033	1.9	6.7	50
2-Chlorophenol	2CLP	0.06	0.99	13	200
1,3-Dichlorobenzene	13DCLB	0.13	1.7	13	200
1,4-Dichlorobenzene	14DCLB	0.098	1.7	13	200
Benzyl alcohol	BZALC	0.19	0.72	13	100
1,2-Dichlorobenzene	12DCLB	0.11	1.7	13	50
2-Methylphenol	2MP	0.029	7.9	1.3	200
Bis(2-chloroisopropyl)ether	B2CIPE	0.2	5.3	13	200
4-Methylphenol	4MP	0.24	0.52	1.3	200
N-Nitroso-di-n-propylamine	NNDNPA	0.2	4.4	13	50
Hexachloroethane	CL6ET	0.15	1.5	13	50
Nitrobenzene	NB	0.045	0.5	13	50
Isophorone	ISOPHR	0.033	4.8	13	50
2-Nitrophenol	2NP	0.14	3.7	13	100
2,4-Dimethylphenol	24DMPN	0.69	5.8	1.3	100
Benzoic acid	BENZOA	6.1*	13.0	--	100
Bis(2-chloroethoxy)methane	B2CEXM	0.059	1.5	13	50
2,4-Dichlorophenol	24DCLP	0.18	2.9	13	200
1,2,4-Trichlorobenzene	124TCB	0.04	1.8	13	50
Naphthalene	NAP	0.039	0.5	3.3	20
4-Chloroaniline	4CANIL	0.81	7.3	3.3	100
Hexachlorobutadiene	HCBD	0.23	3.4	13	100
4-Chloro-3-methylphenol	4CL3C	0.095	4.0	13	200
2-Methylnaphthalene	2MNAP	0.049	1.7	6.7	50
Hexachlorocyclopentadiene	CL6CP	6.2	8.6	13	100
2,4,6-Trichlorophenol	246TCP	0.17	4.2	13	100
2,4,5-Trichlorophenol	245TCP	0.1	5.2	13	200
2-Chloronaphthalene	2CNAP	0.036	0.5	13	200
2-Nitroaniline	2ANIL	0.062	4.3	13	100
Dimethylphthalate	DMP	0.17	1.5	13	100
Acenaphthylene	ANAPYL	0.035	0.5	6.7	50
2,6-Dinitrotoluene	26DNT	0.085	0.79	13	200
3-Nitroaniline	3NANIL	0.45	4.9	13	100

LEGEND:

[1] USATHAMA Method LM18.
[2] USATHAMA Method UM18.
* Quantitation limit for non-certified analyte.

USATHAMA REPORTING LIMITS FOR EPA TCL SEMIVOLATILES (Continued)

ANALYTE	USATHAMA ACRONYM	REPORTING LIMIT SOIL[1] (µg/g)	REPORTING LIMIT WATER[2] (µg/L)	UPPER REPORTING LIMIT SOIL (µg/g)	UPPER REPORTING LIMIT WATER (µg/L)
Acenaphthene	ANAPNE	0.036	1.7	13	50
2,4-Dinitrophenol	24DNP	2.1	21.0	6.7	100
4-Nitrophenol	4NP	1.4	12.0	33	100
Dibenzofuran	FURANS	0.035	1.7	6.7	50
2,4-Dinitrotoluene	24DNT	0.14	4.5	13	200
Diethylphthalate	DEP	0.24	2.0	6.7	200
4-Chlorophenyl-phenylether	4CLPPE	0.033	5.1	13	100
Fluorene	FLRENE	0.033	3.7	13	50
4-Nitroaniline	4NANIL	0.41	5.2	13	100
4,6-Dinitro-2-methylphenol	46DN2C	0.55	17.0	13	100
N-Nitrosodiphenylamine	NNDPA	0.19	3.0	13	200
4-Bromophenyl-phenyl ether	4BRPPE	0.033	4.2	6.7	100
Hexachlorobenzene	CL6BZ	0.033	1.6	6.7	100
Pentachlorphenol	PCP	1.23	18.0	6.7	100
Phenanthrene	PHANTR	0.033	0.5	13	100
Anthracene	ANTRC	0.033	0.5	13	100
Di-n-butylphthalate	DNBP	0.061	3.7	3.3	200
Fluoranthene	FANT	0.068	3.3	13	100
Pyrene	PYR	0.033	2.8	3.3	100
Butylbenzylphthalate	BBZP	0.17	5.4	6.7	50
3,3'-Dichlorobenzidine	33DCBD	6.3	12.0	13	100
Benz[a]anthracene	BAANTR	0.17	1.6	13	100
Chrysene	CHRY	0.12	2.4	6.7	100
Bis(2-ethylhexyl)phthalate	B2EHP	0.62	4.8	13	100
Di-n-octylphthalate	DNOP	0.19	15.0	6.7	100
Benzo[b]fluoranthene	BBFANT	0.21	5.4	3.3	50
Benzo[k]fluoranthene	BKFANT	0.066	0.87	0.67	100
Benzo[a]pyrene	BAPYR	0.25	4.7	13	100
Indeno(1,2,3-cd)pyrene	ICDPR	0.29	8.6	13	100
Dibenz[a,h]anthracene	DBAHA	0.21	6.5	13	50
Benzo[g,h,i]perylene	BGHIPY	0.25	6.1	3.3	50

LEGEND:

1 USATHAMA Method LM18.
2 USATHAMA Method UM18.
* Detection limit for non-certified compounds.

USATHAMA REPORTING LIMITS FOR EPA TCL PESTICIDES/PCBs

ANALYTE	USATHAMA ACRONYM	QUANTITATION LIMIT SOIL[1] (µg/g)	REPORTING LIMIT WATER[2] (µg/L)
alpha-BHC	ABHC	0.27	4.0
beta-BHC	BBHC	0.27	4.0
delta-BHC	DBHC	0.27	4.0
gamma-BHC (Lindane)	LIN	0.27	4.0
Heptachlor	HPLC	0.13	2.0
Aldrin	ALDRN	0.33	4.7
Heptachlor epoxide	HPCLE	0.33	5.0
Endosulfan I	AENSLF	0.62	9.2
Dieldrin	DLDRN	0.31	4.7
4,4'-DDE	PPDDE	0.31	4.7
Endrin	ENDRN	0.45	7.6
Endosulfan II	BENSLF	0.62	9.2
4,4'-DDD	PPDDD	0.30	4.0
Endosulfan sulfate	ESFSO4	0.62	9.2
4,4'-DDT	PDDDT	0.31	9.2
Endrin ketone	ENDRNK	0.53	8.0
Methoxychlor	MEXCLR	0.33	5.1
alpha-Chlordane	ACLDAN	0.33	5.1
gamma-Chlordane	GCLDAN	0.33	5.1
Toxaphene	TXPHEN	2.6	36.0
AROCLOR-1016		1.4	21.0
AROCLOR-1221		1.4	21.0
AROCLOR-1232		1.4	21.0
AROCLOR-1242		1.4	30.0
AROCLOR-1248		2.0	30.0
AROCLOR-1254		2.3	36.0
AROCLOR-1260		2.6	36.0

LEGEND:

1 USATHAMA Method LM18.
2 USATHAMA Method UM18.

NOTE: There are no upper reporting limits for the analytes on this page.

USATHAMA REPORTING LIMITS FOR EPA TCL METALS

ANALYTE	USATHAMA ACRONYM	REPORTING LIMIT		UPPER REPORTING LIMIT	
		SOIL[1] (µg/g)	WATER[2] (µg/L)	SOIL (µg/g)	WATER (µg/L)
Aluminum	AL	14.1	141	5,000	45,000
Antimony	SB	3.8	38	5,000	6,000
Arsenic	AS	0.25	2.54	0.25	10
Barium	BA	29.6	5	200	10,000
Beryllium	BE	1.86	5	20	1,000
Cadmium	CD	3.05	4	20	5,000
Calcium	CA	59.0	500	5,000	20,000
Chromium	CR	12.7	6	5,000	5,000
Cobalt	CO	15.0	25	5,000	500,000
Copper	CU	58.6	8.1	5,000	10,000
Iron	FE	50.0	42.7	5,000	50,000
Lead	PB	0.177	1.26	10	100
Magnesium	MG	50.0	500	5,000	20,000
Manganese	MN	0.275	2.75	5,000	2,000
Mercury	HG	0.05	0.243	1.0	10
Nickel	NI	12.6	34.3	5,000	15,000
Potassium	K	37.5	375	5,000	12,500
Selenium	SE	0.25	3.02	10	100
Silver	AG	2.5	4.6	50	2,500
Sodium	NA	150.0	500	5,000	50,000
Thallium	TL	31.3	81.4	5,000	40,000
Vanadium	V	0.775	3.82	20	200
Zinc	ZN	30.2	21.1	5,000	20,000

LEGEND:

1 USATHAMA Methods JB01, JD15, JD16, JD17, JD18, JD19, and JS11.
2 USATHAMA Methods SB01, SD19, SD20, SD21, SD22, SD23, and SS10.

USATHAMA REPORTING LIMITS FOR EXPLOSIVES

ANALYTE	USATHAMA ACRONYM	REPORTING LIMIT		UPPER REPORTING LIMIT	
		SOIL[1] (µg/g)	WATER[2] (µg/L)	SOIL (µg/g)	WATER (µg/L)
Nitrobenzene	NB	2.41	1.07	27.4	54.9
1,3-Dinitrobenzene	13DNB	0.496	0.519	24.8	40.1
1,3,5-Trinitrobenzene	135TNB	0.448	0.626	24.4	42.1
2,4,6-Trinitrotoluene	246TNT	0.456	0.588	22.8	40.2
2,4-Dinitrotoluene	24DNT	0.424	0.612	21.2	40.2
2,6-Dinitrotoluene	26DNT	0.524	1.15	26.2	52.4
Cyclotetramethylene tetranitramine	HMX	0.666	1.65	33.3	28.9
Cyclotrimethylene-trinitramine	RDX	0.587	2.11	21.9	43.9
N-Methyl-N,2,4,6-tetranitroaniline	TETRYL	0.731	0.556	20.2	44.5

LEGEND:

1 USATHAMA Method LW12.
2 USATHAMA Method UW14.

ANALYTICAL MATRIX REQUEST

SURFACE WATER TAL/TCL AND EXPLOSIVES

SITE ID	FIELD SAMPLE NO.	SAMPLING DATE
RVER-2	RVER-2	8/22/90
RVER-1	RVER-1	12/01/90
CREK-3	CREK-3	8/21/90
DTCHB-1	SDTB-1	8/12/90
DTCHB-3	SDTB-3	8/12/90
DTCHB-2	SDTB-2A	10/8/90
DTCHB-4	SDTB-4A	10/8/90
DTCHC-1	SDTC-1	8/10/90
DTCHC-1	SDTC-3	8/11/90
DTCHC-1	SDTC-1A	10/7/90
DTCHC-3	SDTC-3A	10/7/90
DTCH1-1	SDT1-1	8/13/90
DTCH1-2	SDT1-2	8/13/90
DTCH2-2	SDT2-2	8/10/90
DTCH2-3	SDT2-3	8/14/90
DTCH4-2	SDT4-2	8/22/90
DTCH4-3	SDT4-3	8/22/90
DTCH4-3	SDT4-3	9/13/90
DTCH4-4	SDT4-4	9/13/90
DTCH5-2	SDT5-2	8/14/90
DTCH5-3	SDT5-3	8/15/90
DTCH6-1	SDT6-1	8/24/90
DTCH6-2	SDT6-2	8/24/90
DTCH7-6	SDT7-6	9/13/90
DTCH7-6	SDT7-6	9/13/90
DTCH8-3	SDT8-3	8/20/90
DTCH10-3A	SDT10-3A	9/12/90
DTCH10-6	SDT10-6	9/12/90

SURFACE WATER SELECT METALS AND EXPLOSIVES

SITE ID	FIELD SAMPLE NO.	SAMPLING DATE
DTCHB-2	SDTB-2	8/12/90
DTCHB-4	SDTB-4	8/13/90
DTCHC-2	SDTC-2	8/11/90
DTCHC-2	SDTC-2X	8/11/90
DTCHC-4	SDTC-4	8/13/90

Sediment TAL/TCL and Explosives

Site ID	Field Sample Number	Sampling Date
RVER-1	RVER-1A	8/6/90
RVER-2	RVER-2A	8/22/90
CREK-1	CREK-1A	8/26/90
CREK-2	CREK-2A	8/26/90
CREK-3	CREK-3A	8/21/90
CREK-4	CREK-4A	8/22/90
CREK-5	CREK-5A	8/22/90
DTCHB-1	DTB-1A	8/12/90
DTCHB-2	DTB-2A	8/12/90
DTCHB-3	DTB-3A	8/12/90
DTCHB-4	DTB-4A	8/13/90
DTCHB-1	DTC-1A	8/10/90
DTCHB-2	DTC-2A	8/11/90
DTCHB-3	DTC-3A	8/11/90
DTCHB-4	DTC-4A	8/13/90
DTCHB1-1	DT1-1A	8/7/90
DTCHB1-2	DT1-2A	8/7/90
DTCHB1-3	DT1-3A	8/7/90
DTCHB1-4	DT1-4A	8/7/90
DTCHB1-5	DT1-5A	8/8/90
DTCHB1-6	DT1-6A	8/8/90
DTCHB2-1	DT2-1A	8/9/90
DTCHB2-2	DT2-2A	8/8/90
DTCHB2-3	DT2-3A	8/9/90
DTCHB2-4	DT2-4A	8/9/90
DTCHB2-5	DT2-5A	8/9/90
DTCHB2-6	DT2-6A	8/10/90
DTCHB3-1	DT3-1A	8/23/90
DTCHB3-2	DT3-2A	8/23/90
DTCHB3-3	DT3-3A	8/24/90
DTCHB3-4	DT3-4A	8/24/90
DTCHB3-5	DT3-5A	8/24/90
DTCHB3-6	DT3-6A	8/24/90

Sediment TAL TCL and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
DTCHB4-1	DT4-1A	8/22/90
DTCHB4-2	DT4-2A	8/22/90
DTCHB4-3	DT4-3A	8/22/90
DTCHB4-4	DT4-4A	8/22/90
DTCHB4-5	DT4-5A	8/23/90
DTCHB4-6	DT4-6A	8/23/90
DTCHB5-1	DT5-1A	8/14/90
DTCHB5-2	DT5-2A	8/14/90
DTCHB5-3	DT5-3A	8/15/90
DTCHB5-4	DT54-5A	8/21/90
DTCHB5-5	DT5-5A	8/21/90
DTCHB5-6	DT5-6A	8/21/90
DTCHB6-1	DT6-1A	8/24/90
DTCHB6-2	DT6-2A	8/24/90
DTCHB6-3	DT6-3A	8/25/90
DTCHB6-4	DT6-4A	8/25/90
DTCHB6-5	DT6-5A	8/25/90
DTCHB6-6	DT6-6A	8/25/90
DTCHB7-2	DT7-2A	8/26/90
DTCHB7-3	DT7-3A	8/26/90
DTCHB7-4	DT7-4A	8/26/90
DTCHB7-5	DT7-5A	8/26/90
DTCHB7-6	DT7-6A	8/26/90
DTCHB8-1	DT8-1A	8/20/90
DTCHB8-2	DT8-2A	8/20/90
DTCHB8-3	DT8-3A	8/20/90
DTCHB8-4	DT8-4A	8/21/90
DTCHB8-5	DT8-5A	8/21/90
DTCHB9-2	DT9-2A	8/23/90
DTCHB9-3	DT9-3A	8/23/90
DTCHB9-4	DT9-4A	8/23/90
DTCHB9-5	DT9-5A	8/23/90

Sediment TAL TCL and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
DTCHB9-6	DT9-6A	8/25/90
DTCHB10-1	DT10-1A	8/25/90
DTCHB10-2	DT10-2A	8/26/90
DTCHB10-3	DT10-3A	8/25/90
DTCHB10-4	DT10-4A	8/26/90
DTCHB10-5	DT10-5A	8/26/90
DTCHB10-6	DT10-6A	8/26/90
MI073	MI-SED*172	8/20/90
Drilling Mud	MI-SED*158	8/24/90
MI073	MI-SED*191	8/9/90
MI061	MI-SED*160	8/5/90
MI064	MI-SO*45	8/21/90
MI063	MI-SO*47	8/31/90
MI070	MI-SO*45	10/21/90
MI057	MI-20*48	11/03/90

Sediment Select Metals and Explosives

Site ID	Field Sample Number	Sampling Data
RVER-2	RVER-2B	8/22/90
CREK-1	CREK-1B	8/26/90
CREK-2	CREK-2B	8/26/90
CREK-6	CREK-6A	8/13/90
CREK-6	CREK-6B	8/13/90
CREK-3	CREK-3B	8/21/90
CREK-4	CREK-4B	8/22/90
CREK-5	CREK-5B	8/22/90
DTCHB-1	DTB-1B	8/12/90
DTCHB-1	DTB-1C	8/12/90
DTCHB-2	DTB-2B	8/12/90
DTCHB-2	DTB-2C	8/12/90
DTCHB-3	DTB-3B	8/12/90
DTCHB-3	DTB-3C	8/12/90
DTCHB-4	DTB-4B	8/13/90
DTCHB-4	DTB-4C	8/13/90
DTCHC-1	DTC-1B	8/10/90
DTCHC-1	DTC-1C	8/10/90
DTCHC-2	DTC-2B	8/11/90
DTCHC-2	DTC-2C	8/11/90
DTCHC-3	DTC-3B	8/11/90
DTCHC-3	DTC-3C	8/11/90
DTCHC-4	DTC-4B	8/13/90
DTCHC-4	DTC-4C	8/13/90
DTCH1-1	DT1-1B	8/7/90
DTCH1-2	DT1-2B	8/7/90
DTCH1-3	DT1-3B	8/7/90
DTCH1-4	DT1-4B	8/7/90
DTCH1-6	DT1-6B	8/8/90
DTCH2-1	DT2-1B	8/9/90
DTCH2-2	DT2-2B	8/8/90
DTCH2-3	DT2-3B	8/9/90
DTCH2-4	DT2-4B	8/9/90

Sediment Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
DTCH2-5	DT2-5B	8/9/90
DTCH2-6	DT2-6B	8/10/90
DTCH3-1	DT3-1B	8/23/90
DTCH3-2	DT3-2B	8/23/90
DTCH3-3	DT3-3B	8/24/90
DTCH3-4	DT3-4B	8/24/90
DTCH3-5	DT3-5B	8/24/90
DTCH3-6	DT3-6B	8/24/90
DTCH4-1	DT4-1B	8/22/90
DTCH4-2	DT4-2B	8/22/90
DTCH4-3	DT4-3B	8/22/90
DTCH4-4	DT4-4B	8/22/90
DTCH4-5	DT4-5B	8/23/90
DTCH4-6	DT4-6B	8/23/90
DTCH5-1	DT5-1B	8/14/90
DTCH5-2	DT5-2B	8/14/90
DTCH5-3	DT5-3B	8/15/90
DTCH5-4	DT5-4B	8/21/90
DTCH5-5	DT5-5B	8/21/90
DTCH5-6	DT5-6B	8/21/90
DTCH6-1	DT6-1B	8/24/90
DTCH6-2	DT6-2B	8/24/90
DTCH6-3	DT6-3B	8/25/90
DTCH6-4	DT6-4B	8/25/90
DTCH6-5	DT6-5B	8/25/90
DTCH6-6	DT6-6B	8/25/90
DTCH7-2	DT7-2B	8/26/90
DTCH7-3	DT7-3B	8/26/90
DTCH7-4	DT7-4B	8/26/90
DTCH7-5	DT7-5B	8/26/90
DTCH7-6	DT7-6B	8/26/90
DTCH8-1	DT8-1B	8/20/90

Sediment Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
DTCH8-2	DT8-2B	8/20/90
DTCH8-3	DT8-3B	8/20/90
DTCH8-4	DT8-4B	8/21/90
DTCH8-5	DT8-5B	8/21/90
DTCH9-2	DT9-2B	8/23/90
DTCH9-3	DT9-3B	8/23/90
DTCH9-4	DT9-4B	8/23/90
DTCH9-5	DT9-5B	8/23/90
DTCH9-6	DT9-6B	8/25/90
DTCH10-1	DT10-1B	8/25/90
DTCH10-2	DT10-2B	8/26/90
DTCH10-3	DT10-3BR	8/12/90
DTCH10-4	DT10-4B	8/26/90
DTCH10-5	DT10-5B	8/26/90
DTCH10-6	DT10-6B	8/26/90

Groundwater Select Metals and Explosives

Site ID	Field Sample Number	Sampling Date
MI001	MI001	10/21/90
MI002	MI002	10/30/90
MI003	MI003	10/17/90
MI004	MI004	11/12/90
MI005	MI005	10/16/90
MI007	MI007	11/01/90
MI008	MI008	10/31/90
MI010	MI010	11/10/90
MI011	MI011	11/01/90
MI012	MI012	11/04/90
MI013	MI013	11/05/90
MI015	MI015	11/10/90
MI016	MI016	11/01/90
MI018	MI018	11/05/90
MI019	MI019	11/06/90
MI020	MI020	11/10/90
MI021	MI021	11/13/90
MI022	MI022	11/09/90
MI023	MI023	10/31/90
MI024	MI024	11/09/90
MI025	MI025	11/08/90
MI026	MI026	11/04/90
MI027	MI027	11/09/90
MI028	MI028	11/07/90
MI029	MI029	11/11/90
MI031	MI031	11/12/90
MI032	MI032	11/12/90
MI033	MI033	11/07/90
MI036	MI036	10/28/90
MI037	MI037	10/30/90
MI038	MI038	10/25/90
MI039	MI039	10/24/90
MI040	MI040	10/26/90

Groundwater Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
MI041	MI041	10/27/90
MI044	MI044	11/02/90
MI045	MI045	11/02/90
MI046	MI046	10/21/90
MI047	MI047	10/23/90
MI048	MI048	10/30/90
MI052	MI052	10/31/90
MI053	MI053	10/31/90
MI056	MI056	11/05/90
MI058	MI058	11/02/90
MI059	MI059	11/04/90
MI061	MI061	11/08/90
MI062	MI062	11/03/90
MI064	MI064	11/06/90
MI065	MI065	11/04/90
MI066	MI066	12/12/90
MI067	MI067	12/12/90
MI068	MI068	11/06/90
MI069	MI069	11/29/90
MI070	MI070	12/02/90
MI074	MI074	11/03/90
MI075	MI075	11/30/90
MI076	MI076	11/08/90
MI077	MI077	11/09/90
MI078	MI078	11/29/90
MI079	MI079	11/29/90
MI080	MI080	11/29/90
MI081	MI081	11/28/90
MI082	MI082	11/28/90
001	001	11/06/90
002	002	11/06/90
003	003	11/07/90

Groundwater Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
004	004	11/04/90
005	005	11/07/90
006	006	11/04/90
007	007	11/08/90
K-100	K-100	10/18/90
T-100	T-100	10/18/90
F-100	F-100	10/19/90
X-100	X-100	10/19/90
Y-100	Y-100	10/19/90
P-97	P-97	10/19/90
ZZ-3	ZZ-3	10/19/90
K-323	K-323	10/19/90
E-67	E-67	10/18/90
C-5	C-5	10/18/90
I-11	I-11	10/18/90
T-99	T-99	10/18/90
S-99	S99	10/19/90
DW001	DW001	11/27/90
DW002	DW002	11/27/90

Groundwater TAL/TCL and Explosives

Site ID	Field Sample Number	Sampling Date
MI006	MI006	10/16/90
MI009	MI009	11/03/90
MI014	MI014	11/11/90
MI017	MI017	11/07/90
MI030	MI030	11/11/90
MI034	MI034	10/15/90
MI035	MI035	10/28/90
MI049	MI049	11/05/90
MI050	MI050	11/01/90
MI051	MI051	10/21/90
MI054	MI054	10/29/90
MI055	MI055	10/22/90
MI057	MI057	11/29/90
MI060	MI060	11/30/90
MI063	MI063	11/03/90
MI071	MI071	11/01/90
MI072	MI072	11/03/90
MI073	MI073	11/02/90

Soil Borings Select Metals and Explosives

Site ID	Field Sample Number	Sampling Data
OBGA-1	OBGA-1B	09/08/90
OBGA-1	OBGA-1C	09/08/90
OBGA-1	OBGA-1D	09/08/90
OBGA-1	OBGA-1E	09/08/90
OBGA-1S	OBGA-1F	09/08/90
OBGA-2	OBGA-2B	09/07/90
OBGA-2	OBGXA-2B	09/07/90
OBGA-2S	OBGA-2C	09/07/90
OBGA-2	OBGA-2D	09/07/90
OBGA-2	OBGA-2E	09/07/90
OBGA-3	OBGA-3A	08/12/90
OBGA-3	OBGA-3C	08/12/90
OBGA-3	OBGA-3D	08/12/90
OBGA-3S	OBGA-3E	08/13/90
OBGA-4	OBGA-4B	08/29/90
OBGA-4	OBGA-4C	08/29/90
OBGA-4	OBGA-4D	09/05/90
OBGA-4S	OBGA-4E	09/05/90
OBGA-4	OBGA-4G	09/06/90
OBGA-4	OBGA-4G	09/06/90
OBGA-5S	OBGA*17	07/31/90
OBGA-5	OBGA*13	07/31/90
OBGA-6	OBGA*1	07/26/90
OBGA-6	OBGA*2	07/27/90
OBGA-6	OBGA*3	07/27/90
OBGA-6	OBGA*4	07/27/90
OBGA-2S	OBGA-2E	08/27/90
OBGA-2	OBGA-2B	08/27/90
OBGA-2	OBGA-2C	08/27/90
OBGA-2	OBGA-2D	08/27/90
OBGA-5	OBGA-5A	08/14/90

Soil Borings Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
OBGA-5	OBGA-5C	08/14/90
OBGA-5	OBGA-5D	08/14/90
OBGA-5S	OBGA-5E	08/14/90
OBGA-3S	OBGA-3A	08/07/90
OBGA-3	OBGA-3C	08/08/90
OBGA-3	OBGA-3D	08/08/90
OBGA-4	OBGA-4B	08/08/90
OBGA-4	OBGA-4C	08/08/90
ADAB-1	ADAB-1B	08/25/90
ADAB-1	ADAB-1C	08/25/90
ADAB-1S	ADAB-1D	08/25/90
ADAB-1	ADAB-1E	08/25/90
ADAB-1	ADAXB-1E	08/25/90
ADAB-2	ADAB-2A	08/26/90
ADAB-2	ADAXB-2A	08/26/90
ADAB-2	ADAB-2C	08/26/90
ADAB-2S	ADAB-2D	08/26/90
ADAB-2	ADAB-2E	08/27/90
CBG-1	CBG-1B	09/08/90
CBG-1	CBG-1C	09/08/90
CBG-2	CBG-2A	09/08/90
CBG-2	CBG-2B	09/08/90
CBG-3	CBG-3A	09/09/90
CBG-3	CBG-3B	09/09/90
CBG-4	CBG-4A	09/09/90
CBG-4	CBG-4C	09/09/90
CBG-4	CBGX-4C	09/09/90
CBG-5	CBG-5A	09/09/90
CBG-5	CBG-5B	09/09/90
OBGB-3	OBGB-3B	08/28/90
OBGB-3	OBGB-3C	08/28/90

Soil Borings Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
OBGB-3	OBGXB-3C	08/28/90
OBGB-3	OBGB-3D	08/28/90
OBGB-3S	OBGB-3E	08/28/90
OBGB-4	OBGB-4B	08/23/90
OBGB-4	OBGB-4C	08/23/90
OBGB-4	OBGXB-4C	08/23/90
OBGB-4S	OBGB-4D	08/23/90
OBGB-4	OBGB-4E	08/24/90
OBGB-5	OBGB-5A	08/21/90
OBGB-5	OBGXB-5A	08/21/90
OBGB-5	OBGB-5B	08/21/90
OBGB-5	OBGB-5L	08/21/90
OBGB-5S	OBGB-5D	08/21/90
OBGB-6	OBGB*6	07/28/90
OBGB-6	OBGB*7	07/29/90
OBGB-6S	OBGB*9	07/29/90
OBGB-6	OBGB*10	07/30/90
OBGC-2S	OBGB*15	08/01/90
OBGC-3	OBGB-3A	08/09/90
OBGC-3	OBGB-3C	08/09/90
OBGC-3	OBGXB-3C	08/09/90
OBGC-3S	OBGB-3D	08/09/90
OBGC-3	OBGB-3E	08/10/90
OBGC-4	OBGB-4A	08/20/90
OBGC-4	OBGB-4B	08/20/90
OBGC-4	OBGB-4C	08/20/90
OBGC-4S	OBGB-4E	08/20/90
CDP-1	CDP-1A	09/11/90
CDP-1	CDPX-1A	09/11/90
CDP-1	CDP-1C	09/11/90
CDP-1S	CDP-1D	09/11/90

Soil Borings Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
CDP-1	CDP-1E	09/11/90
CDP-2	CDP-2B	09/09/90
CDP-2	CDP-2C	09/09/90
CDP-2	CDP-2D	09/09/90
CDP-2	CDP-2E	09/09/90
CDP-3	CDP-3A	09/12/90
CDP-3	CDP-3C	09/12/90
CDP-3	CDP-3E	09/12/90
CDP-3	CDP-3F	09/12/90
CLF-1	CLF-1A	09/11/90
CLF-1	CLF-1B	09/11/90
CLF-2	CLF-2A	09/11/90
CLF-2	CLF-2C	09/11/90
CLF-3	CLF-3A	09/12/90
CLF-3	CLF-3B	09/12/90
CLF-4	CLF-4A	09/13/90
CLF-4	CLFX-4A	09/13/90
CLF-4	CLF-4C	09/13/90
CLF-5	CLF-5B	09/13/90
CLF-5	CLF-5C	09/13/90
LF-1	LF-1A	09/13/90
LF-1	LF-1C	09/13/90
LF-2	LF-2A	09/13/90
LF-2	LFX-2A	09/13/90
SA-7	SA-7A	10/04/90
SA-7	SA-7C	10/04/90
SA-44	SA-44A	10/04/90
SA44	SA-44C	10/04/90
SA-40	SA-40A	10/05/90
SA-40	SA-40C	10/05/90
SA-4	SA-4A	11/08/90

Soil Borings Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
SA-4	SA-4C	10/08/90
SB-18	SB-18A	09/25/90
SB-18	SB-18C	09/25/90
SB-18	SBX-18C	09/25/90
SB-12	SB-12A	11/02/90
SB-12	SB-12B	11/02/90
SB-273	SB-273A	10/26/90
SB-273	SB-273C	10/26/90
SB-10	SB-10A	10/05/90
SB-10	SB-10B	10/05/90
SB-2	SB-2A	10/05/90
SB-2	SB-2B	10/05/90
SC-42	SC-42B	09/13/90
SC-42	SC-42C	09/13/90
SC-12	SC-12A	09/19/90
SC-12	SC-12C	09/19/90
SC-5	SC-5A	09/19/90
SC-5	SC-5C	09/19/90
SC-5	SCX-5C	09/19/90
SC-42E	SC-42EA	09/20/90
SC-42E	SC-42FC	09/20/90
SC-1	SC-1A	09/20/90
SC-1	SC-1C	09/20/90
SC-6	SC-6B	09/14/90
SC-6	SC-6C	09/14/90
SD-10	SD-10A	09/22/90
SD-10	SD-10C	09/22/90
SD-42B	SD-42B-B	10/09/90
SD-42B	SD-42B-C	10/09/90
SE-4	SE-4A	09/26/90
SE-4	SE-4C	09/26/90

Soil Borings Select Metals and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Data
SE-4	SEX-4C	09/26/90
S0-3	S0-3A	10/11/90
S0-3	S0-3C	10/11/90
S0-14	S0-14A	09/25/90
S0-14	S0-14C	09/25/90
SX-8	SX-8A	10/03/90
SX-8	SX-8C	10/03/90
SX-313	SX-313B	10/25/90
SX-313	SX-313B	10/25/90
SX-313	SX-131C	10/25/90
SX-103	SX-103A	10/26/90
SX-103	SX-103B	10/26/90
SX-41	SX-41A	11/05/90
SX-41	SX-41B	11/05/90
SX-26	SX-26A	11/09/90
SX-26	SX-26B	11/09/90
SZ-4	SZ-4A	09/20/90
SZ-4	SZ-4C	09/20/90
SZ-4W	SZ-4WA	09/22/90
SZ-4W	SZ-4WB	09/22/90
SZ-2	SZ-2B	09/22/90
SZ-2	SZ-2C	09/22/90
SZ-2	SZ-2C	09/22/90

Soil Borings
TAL/TCL and Explosives

Site ID	Field Sample Number	Sampling Date
CLF-1	CLF-1C	9/11/90
CLF-2	CLF-2B	9/11/90
CLF-3	CLF-3C	9/12/90
CLF-3	CLFX-3C	9/12/90
CLF-4	CLF-4B	9/13/90
CLF-5	CLF-5A	9/13/90
LF-1	LF-1B	9/13/90
LF-1	LFX-1B	9/13/90
LF-2	LF-2C	9/13/90
SYD-1	SYD-1A	9/12/90
SYD-1	SYDX-1A	9/12/90
SYD-2	SYD-2A	9/12/90
SA-7	SA-7B	10/4/90
SA-7	SA-X7B	10/4/90
SA-44	SA-44B	10/4/90
SA-A44	SA-A44	10/9/90
SA-4D	SA-40B	11/5/90
SA-4	SA-4B	11/8/90
SB-18	SB-18B	9/25/90
SB-12	SB-12C	11/2/90
SB-273	SB-273B	10/26/9
SB-A10	SB-A10	10/7/90
SB-10	SB-10C	10/5/90
SB-2	SB-2C	10/5/90
SC-A42E	SC-A42E	10/8/90
SC-42	SC-42A	9/13/90
SC-12	SC-12B	9/19/90
SC-5	SC-5B	9/19/90
SC-42E	SC-42EB	9/20/90
SC-42E	SCX-42EB	9/20/90

Soil Borings
TAL/TCL and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
SC-1	SC-1B	9/20/90
SC-6	SC-6	9/14/90
SD-10	SD-10B	9/22/90
SD-42B	SD-42B-A	10/9/90
SD-41	SD-41A	10/10/9
OBGA-1	OBGA-1A	9/8/90
OBGA-2	OBGA-2A	9/7/90
OBGA-2	OBGA-2A	9/7/90
OBGA-3	OBGA-3B	8/12/90
OBGA-4	OBGA-4A	8/29/90
OBGA-4	OBGXA-4A	8/29/90
OBGA-4S	OBGA-4F	9/6/90
OBGA-4S	OBGXA-4F	9/6/90
OBGA-5	OBG*12	7/31/90
OBGB-2	OBGB-2A	8/27/90
OBGB-3	OBGB-3A	8/28/90
OBGB-4	OBGB-4A	8/23/90
OBGB-5	OBGB-5E	8/22/90
OBGB-6	OBG*5	7/28/90
OBGC-2	OBG*14	7/31/90
OBGC-3	OBGC-3B	8/9/90
OBGC-4	OBGC-4D	8/20/90
OBGC-5	OBGC-5B	8/14/90
OBGD-3	OBGD-3B	8/7/90
OBGD-4S	OBGD-4A	8/8/90
ADAB-1	ADAB-1A	8/25/90
ADAB-2	ADAB-2B	8/26/90
CBG-1	CBG-1A	9/8/90
CBG-2	CBG-2C	9/8/90

Soil Borings
TAL/TCL and Explosives
(Continued)

Site ID	Field Sample Number	Sampling Date
CBG-3	CBG-3C	9/9/90
CBG-3	CBGX-3C	9/9/90
CBG-4	CBG-4B	9/9/90
CBG-5	CBG-5C	9/9/90
CDP-1	CDP-1B	9/11/90
CDP-1	CDPX-1B	9/11/90
CDP-2	CDP-2A	9/9/90
CDP-3	CDP-3B	9/12/90
CDP-3	CDP-3D	9/12/90
SD-42	SD-42A	10/11/9
SD-42	SD-X42A	10/11/9
SE-4	SE-4B	9/26/90
SO-3	SO-3B	10/11/9
SO-14	SO-14B	9/25/90
SO-14	SOX-14B	9/25/90
SX-8	SX-8B	10/3/90
SX-313	SX-313A	10/25/9
SX-103	SX-103C	10/26/9
SX-41	SX-41C	11/5/90
SX-26	SX-26C	11/9/90
SX-26	SZ-4B	9/20/90
SZ-4	SZ-4WC	9/22/90
SZ-4W	SZX-4WC	9/22/90
SZ-4W	SZ-2A	9/22/90
SZ-Z	SZ-A4	10/8/90
SZ-A4	SX-X26C	11/9/90

ESE LOT DESIGNATIONS

Lot Number	Site ID (x) = Multiple Depth Location
PMZ	DTCH1-1 (2), DTCH1-2 (2), DTCH1-3 (2), DTCH1-4 (2), RVER-1
PSH	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, RVER-1
PSM	DTCH1-5, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5
PSN	DTCH2-6, DTCH5-1, DTCH5-2, DTCHB-1, DTCHB-2, DTCHB-3, DTCHB-4, DTCHC-1, DTCHC-2, DTCHC-3, DTCHC-4
PSO	DTCH3-1, DTCH3-2, DTCH3-4, DTCH3-5, DTCH4-5, DTCH4-6, DTCH6-1, DTCH6-2, DTCH9-2, DTCH9-3, DTCH9-4, MI061, MI073
PSQ	CREK-3, CREK-4, CREK-5, DTCH4-2, DTCH4-3, DTCH4-4, DTCH5-6, DTCH8-1, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, RVER-2
PSS	DTCH10-1, DTCH10-3, DTCH6-3
PSV	CREK-2, DTCH10-6, DTCH7-2, DTCH7-4, MI063, MI072 (3)
PSX	MI070
PTI	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, RVER-1
PTN	DTCH1-5, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5
PTO	DTCH2-6, DTCH5-1, DTCH5-2, DTCH5-3, DTCH5-4, DTCHB-1, DTCHB-2, DTCHB-3, DTCHB-4, DTCHC-1, DTCHC-2, DTCHC-3, DTCHC-4
PTP	DTCH3-1, DTCH3-2, DTCH3-3, DTCH3-4, DTCH3-5, DTCH3-6, DTCH4-5, DTCH4-6, DTCH6-1, DTCH6-2, DTCH9-2, DTCH9-3, DTCH9-4, DTCH9-5, MI061, MI073
PTR	CREK-3, CREK-4, CREK-5, DTCH4-1, DTCH4-2, DTCH4-3, DTCH4-4, DTCH5-5, DTCH5-6, DTCH8-1, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, RVER-2
PTT	DTCH10-1, DTCH10-3, DTCH6-3, DTCH6-4, DTCH6-5, DTCH6-6, DTCH9-6
PTW	CREK-1, CREK-2, DTCH10-2, DTCH10-5, DTCH10-6, DTCH7-2, DTCH7-3, DTCH7-4, DTCH7-5, DTCH7-6, MI063, MI072 (3)
RXT	DTCH1-1 (2), DTCH1-2 (2), DTCH1-3 (2), DTCH1-4 (2), RVER-1
RXY	DTCH1-5, DTCH1-6 (2), DTCH2-1 (2), DTCH2-2 (2), DTCH2-3 (2), DTCH2-4 (2), DTCH2-5 (2)

Lot Number	Site ID (x) = Multiple Depth Location
RXZ	DTCH2-6 (2), DTCH5-1 (2), DTCH5-2 (2), DTCH5-3 (2), DTCH5-4 (2), DTCHB-1 (3), DTCHB-2 (3), DTCHB-3 (3), DTCHB-4 (3), DTCHC-1 (3), DTCHC-2 (3), DTCHC-3 (3), DTCHC-4 (3)
SFM	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, RVER-1
SFU	DTCH1-5, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5
SFV	DTCH2-6, DTCH5-1, DTCH5-2, DTCH5-3, DTCH5-4, DTCHB-1, DTCHB-2, DTCHB-3, DTCHB-4, DTCHC-1, DTCHC-2, DTCHC-3, DTCHC-4
SFW	DTCH3-1, DTCH3-2, DTCH3-3, DTCH3-4, DTCH3-5, DTCH3-6, DTCH4-5, DTCH4-6, DTCH6-1, DTCH6-2, DTCH9-2, DTCH9-3, DTCH9-4, DTCH9-5, MI061, MI073
SFY	CREK-3, CREK-4, CREK-5, DTCH4-1, DTCH4-2, DTCH4-3, DTCH4-4, DTCH5-5, DTCH5-6, DTCH8-1, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, RVER-2
SSN	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, DTCH1-5, DTCH1-6, DTCH2-2, RVER-1
SSO	DTCH2-1, DTCH2-3, DTCH2-4, DTCH2-5, DTCH2-6, DTCHC-1
SSP	DTCHB-1, DTCHB-2, DTCHB-3, DTCHB-4, DTCHC-2, DTCHC-3, DTCHC-4
SSQ	DTCH5-1, DTCH5-2, DTCH5-3
SSS	CREK-3, DTCH5-4, DTCH5-5, DTCH5-6, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, MI073
SSU	CREK-4, CREK-5, DTCH4-1, DTCH4-2, DTCH4-3, DTCH4-4, DTCH8-1, RVER-2
SSV	DTCH3-1, DTCH3-2, DTCH3-3, DTCH3-4, DTCH3-5, DTCH3-6, DTCH4-5, DTCH4-6, DTCH6-1, DTCH6-2, DTCH6-3, DTCH6-4, DTCH9-2, DTCH9-3, DTCH9-4, DTCH9-5, MI061
SSW	DTCH10-1, DTCH10-3, DTCH6-5, DTCH6-6, DTCH7-2, DTCH7-3, DTCH9-6
SSY	CREK-1, CREK-2, DTCH10-2, DTCH10-4, DTCH10-5, DTCH7-4, DTCH7-5, DTCH7-6, MI072 (3)
SSZ	DTCH10-6
SUF	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, RVER-1
SUK	DTCH1-5, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5

Lot Number	Site ID (x) = Multiple Depth Location
SUL	DTCH2-6, DTCH5-1, DTCH5-2, DTCH5-3, DTCH5-4, DTCHB-1, DTCHB-2, DTCHB-3, DTCHB-4, DTCHC-1, DTCHC-2, DTCHC-3, DTCHC-4
SUM	DTCH3-1, DTCH3-2, DTCH3-3, DTCH3-4, DTCH3-5, DTCH3-6, DTCH4-5, DTCH4-6, DTCH6-1, DTCH6-2, DTCH9-2, DTCH9-3, DTCH9-4, DTCH9-5, MI061, MI073
SUO	CREK-3, CREK-4, CREK-5, DTCH4-1, DTCH4-2, DTCH4-3, DTCH4-4, DTCH5-5, DTCH5-6, DTCH8-1, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, RVER-2
SUQ	DTCH10-1, DTCH10-3, DTCH6-3, DTCH6-4, DTCH6-5, DTCH6-6, DTCH9-6
SUT	CREK-1, CREK-2, DTCH10-2, DTCH10-4, DTCH10-5, DTCH10-6, DTCH7-2, DTCH7-3, DTCH7-4, DTCH7-5, DTCH7-6, MI063, MI072 (3)
SVJ	DTCH1-1, DTCH1-2, DTCH1-3, RVER-1
SVK	DTCH1-4
SVL	DTCH1-5, DTCH1-6, DTCH2-2
SVM	DTCH2-1, DTCH2-3, DTCH2-4, DTCH2-5, DTCH2-6, DTCHC-1, DTCHC-2, DTCHC-3
SVN	DTCHB-1, DTCHB-3, DTCHC-4
SVO	DTCH5-1, DTCH5-2, DTCH5-3, DTCHB-2, DTCHB-4
SVR	DTCH3-2, DTCH9-2, DTCH9-3, DTCH9-5
SVS	CREK-3, CREK-4, DTCH4-1, DTCH4-2, DTCH4-3, DTCH4-4, DTCH5-4, DTCH5-6, DTCH8-1, DTCH8-2, DTCH8-3, DTCH8-4, DTCH8-5, MI073, RVER-2
SVU	CREK-5, DTCH4-4, DTCH5-5
SVV	DTCH4-5, DTCH4-6, DTCH9-4
SVW	DTCH10-1, DTCH10-3, DTCH3-1, DTCH3-3, DTCH3-4, DTCH3-5, DTCH3-6, DTCH6-1, DTCH6-2, DTCH6-5, DTCH6-6, DTCH9-3, DTCH9-6, MI061
SVX	DTCH6-3, DTCH6-4, DTCH7-3, DTCH7-4
SVY	CREK-1, CREK-2, DTCH10-2, DTCH10-4, DTCH10-5, DTCH10-6, DTCH7-2, DTCH7-3, DTCH7-5, DTCH7-6, MI072 (3)
SWM	DTCH1-1 (2), DTCH1-2 (2), DTCH1-3 (2), DTCH1-4 (2), RVER-1
SWN	DTCH1-5, DTCH1-6 (2), DTCH2-2 (2)

Lot Number	Site ID (x) = Multiple Depth Location
SWO	DTCH2-1 (2), DTCH2-3 (2), DTCH2-4 (2), DTCH2-5 (2)
SWQ	DTCH2-6 (2), DTCHC-1 (3)
SWR	DTCHB-1 (3), DTCHB-2 (3), DTCHB-3 (3), DTCHB-4 (3), DTCHC-2 (3), DTCHC-3 (3), DTCHC-4 (3)
SWT	DTCH5-1 (2), DTCH5-2 (2)
SWU	DTCH5-3 (2)
SWV	DTCH8-1 (2), DTCH8-2 (2), DTCH8-3 (2), MI073
SWW	CREK-3 (2), DTCH5-4 (2), DTCH5-5 (2), DTCH5-6 (2), DTCH8-4 (2), DTCH8-5 (2)
SWX	CREK-4 (2), CREK-5 (2), DTCH4-1 (2), DTCH4-2 (2), DTCH4-3 (2), DTCH4-4 (2), RVER-2 (2)
SWZ	DTCH3-1 (2), DTCH3-2 (2), DTCH4-5 (2), DTCH4-6 (2), DTCH9-2 (2), DTCH9-3 (2), DTCH9-4 (2), DTCH9-5 (2)
TIE	DTCH1-5, DTCH1-6 (2), DTCH2-1 (2), DTCH2-2 (2), DTCH2-3 (2), DTCH2-4 (2), DTCH2-5 (2)
TIF	DTCH2-6 (2), DTCH5-1 (2), DTCH5-2 (2), DTCH5-3 (2), DTCH5-4, DTCHB-1 (3), DTCHB-2 (3), DTCHB-3 (3), DTCHB-4 (3), DTCHC-1 (3), DTCHC-2 (3), DTCHC-3 (3), DTCHC-4 (3)
TIG	CREK-5, DTCH3-1 (2), DTCH3-2 (2), DTCH3-3 (2), DTCH3-4 (2), DTCH3-5 (2), DTCH3-6 (2), DTCH4-5 (2), DTCH4-6 (2), DTCH6-1 (2), DTCH6-2 (2), DTCH9-2 (2), DTCH9-3 (2), DTCH9-5, MI061, MI073
TII	CREK-3 (2), CREK-4, CREK-5, DTCH4-1, DTCH4-2 (2), DTCH4-3 (2), DTCH4-4, DTCH5-5, DTCH8-1 (2), DTCH8-2, DTCH8-3, DTCH8-4 (2), DTCH8-5, RVER-2 (2)
TIK	DTCH10-1 (2), DTCH10-3, DTCH6-3 (2), DTCH6-4 (2), DTCH6-5 (2), DTCH6-6 (2), DTCH9-6 (2)
TIN	CREK-1, CREK-2 (2), CREK-6 (2), DTCH10-3, DTCH10-4 (2), DTCH10-5 (2), DTCH10-6 (2), DTCH7-2 (2), DTCH7-3 (2), DTCH7-4 (2), DTCH7-5, MI063, MI072 (3)
TIW	MI064, MI070
TJB	DTCH3-3 (2), DTCH3-4 (2), DTCH3-5, DTCH3-6 (2), DTCH6-1 (2), DTCH6-2 (2), MI061
TJC	DTCH10-1 (2), DTCH10-3, DTCH6-3 (2), DTCH6-4 (2), DTCH6-5 (2), DTCH6-6 (2), DTCH7-2 (2), DTCH7-3 (2), DTCH7-4 (2), DTCH9-6 (2), MI072 (3)
TJD	CREK-1 (2), CREK-2 (2), DTCH10-2 (2), DTCH10-4 (2), DTCH10-5 (2), DTCH10-6 (2), DTCH7-5 (2), DTCH7-6 (2)

Lot Number	Site ID (x) = Multiple Depth Location
TJN	DTCH10-3
TJO	CREK-6 (2)
TKB	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5, DTCH2-6
TKC	RVER-1
TKE	DTCHB-1, DTCHB-2, DTCHB-3
TKF	DTCH1-1, DTCH1-2, DTCH1-3, DTCH1-4, DTCH1-5, DTCH1-6, DTCH2-1, DTCH2-2, DTCH2-3, DTCH2-4, DTCH2-5, DTCH2-6, DTCHC-1 (3), DTCHC-2 (3), DTCHC-3 (3)
TKJ	DTCH5-1 (2), DTCH5-2 (2), DTCH5-3 (2), DTCHB-1 (2), DTCHB-2 (2), DTCHB-3 (2), DTCHB-4 (3), DTCHC-4 (3)
TKK	CREK-3 (2), DTCH5-4 (2), DTCH5-5 (2), DTCH5-6 (2), DTCH8-1 (2), DTCH8-2 (2), DTCH8-3 (2), DTCH8-4 (2), DTCH8-5 (2), MI073
TKN	CREK-4 (2), CREK-5 (2), DTCH3-1, DTCH4-1 (2), DTCH4-2 (2), DTCH4-3 (2), DTCH4-4 (2), DTCH4-5 (2), DTCH4-6 (2), DTCH9-2 (2), DTCH9-3 (2), DTCH9-4 (2), DTCH9-5 (2), RVER-2 (2)
TKO	DTCH10-1 (2), DTCH10-3, DTCH3-1, DTCH3-2 (2), DTCH3-3 (2), DTCH3-4 (2), DTCH3-5, DTCH3-6 (2), DTCH6-1 (2), DTCH6-2 (2), DTCH6-3 (2), DTCH6-4 (2), DTCH6-5 (2), DTCH6-6 (2), DTCH7-2 (2), DTCH7-3, DTCH9-6 (2)
TKP	DTCH7-3 (2), DTCH7-4 (2), MI061
TKQ	CREK-1 (2), CREK-2 (2), DTCH10-2 (2), DTCH10-4 (2), DTCH10-5 (2), DTCH10-6 (2), DTCH7-5 (2), DTCH7-6 (2), MI072 (3)
TKT	MI063
TKZ	CREK-6 (2), DTCH10-3, MI064
TMC	CREK-3 (2), CREK-4 (2), CREK-5, DTCH4-1 (2), DTCH4-2 (2), DTCH4-3 (2), DTCH4-4 (2), DTCH5-5 (2), DTCH5-6 (2), DTCH8-1 (2), DTCH8-2 (2), DTCH8-3 (2), DTCH8-4 (2), DTCH8-5 (2), RVER-2 (2)
TME	CREK-5, DTCH3-1 (2), DTCH3-2 (2), DTCH3-3 (2), DTCH3-4 (2), DTCH3-5, DTCH3-6 (2), DTCH4-5 (2), DTCH4-6 (2), DTCH6-1 (2), DTCH6-2 (2), DTCH9-2 (2), DTCH9-3 (2), DTCH9-4 (2), DTCH9-5 (2), MI061, MI073
TMH	DTCH10-1 (2), DTCH10-3, DTCH6-3 (2), DTCH6-4 (2), DTCH6-5 (2), DTCH6-6 (2), DTCH9-6 (2)

Lot Number	Site ID (x) = Multiple Depth Location
TMI	CREK-1 (2), CREK-2 (2), DTCH10-2 (2), DTCH10-4 (2), DTCH10-5 (2), DTCH10-6 (2), DTCH7-2 (2), DTCH7-3 (2), DTCH7-4 (2), DTCH7-5 (2), DTCH7-6 (2), MI072 (3)
TMN	CREK-6 (2), DTCH10-3, MI064
TMT	MI063
TMW	MI070
TND	MI064
TNU	MI063
TNZ	MI070
TQK	MI064
TWA	DTCH10-1, DTCH10-3, DTCH6-3, DTCH6-4, DTCH6-5, DTCH6-6, DTCH9-6
TWD	CREK-1, CREK-2, DTCH10-2, DTCH10-4, DTCH10-5, DTCH10-6, DTCH7-2, DTCH7-3, DTCH7-4, DTCH7-5, DTCH7-6, MI063, MI072 (3)
TWM	MI064, MI070
TXG	MI063
TXM	MI070
TYD	MI063
TYI	MI070
UAG	MI070
UGC	MI064, MI070
UGN	DTCH10-4
UHC	MI064, MI070
UME	MI064

SURFACE WATER DUPLICATE ASSESSMENT

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
DTCHC-2	HMX	3.40	3.70	8.45
	RDX	15.3	14.7	-4.00
DTCH7-6	Aluminum	2,270	2,300	1.31
	Barium	96.3	98.1	1.85
	Calcium	6,750	6,920	2.49
	Iron	1,610	1,610	0.00
	Lead	1.41	7.05	133.33
	Magnesium	2,280	2,320	1.74
	Manganese	105	109	3.74
	Potassium	12,900	13,200	2.30
	Sodium	723	776	7.07
	Vanadium	3.82 U	4.62	18.96
	Zinc	24.4	31.1	24.14
	2CHE10	5.00	5.00	0.00

GROUNDWATER DUPLICATE ASSESSMENT

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
MI016U	Lead	12.6	15.2	18.71
	246TNT	6.87	7.5	8.77
MI018	Lead	3.69	6.81	59.43
MI018U	Lead	10.39	13.4	25.30
MI021U	Lead	6.94	7.92	13.19
	Chromium	40.6	42.0	3.39
MI056U	Lead	7.7	9.44	20.30
MI056U	Cadmium	12.1	15.1	22.06
MI056	Cadmium	5.86	6.26	6.60
MI068U	Lead	12.7	14.9	15.94
MI068U	Cadmium	15.2	18.5	19.58
MI068U	Chromium	11.2	11.4	1.77
MI068	Lead	1.74	13.1	153.10
MI081U	Lead	2.39	2.82	16.51
MI082U	Lead	2.49	5.64	77.49
MI082U	Chromium	10.0	13.0	26.09
MI082	Lead	1.41	8.67	144.05

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
MI014U	Mercury	0.265	0.416	44.35
	Vanadium	240	400	50.00
	Lead	47.5	60.0	23.26
	Arsenic	9.91	13.6	31.39
	Silver	0.255	0.302	16.88
	Aluminum	200,000	280,000	33.33
	Barium	516	597	14.56
	Calcium	13,600	16,300	18.06
	Cobalt	48.4	62.8	25.90
	Chromium	133	178	28.94
	Copper	107	111	3.67
	Iron	150,000	210,000	33.33
	Potassium	6,500	7,990	20.57
	Magnesium	9,560	11,900	21.81
	Manganese	1,320	1,610	19.80
	Sodium	7,200	7,840	8.51
	Nickel	90.1	111	20.79
	Zinc	259	282	8.50
MI014	c16	9.0	10.0	10.53
	c17	30.0	30.0	0.00
	c18	30.0	40.0	28.57
	c20	10.0	10.0	0.00

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
MI030U	Lead	1.41	1.84	26.46
	Barium	41.0	43.3	5.46
	Calcium	7,550	8,630	13.35
	Iron	369	531	36.00
	Potassium	604	936	43.12
	Magnesium	1,590	1,770	10.71
	Manganese	36.9	40.6	9.55
	Sodium	7,120	7,360	3.31
MI030	Barium	29.3	29.9	2.03
	Calcium	4,900	5,160	5.17
	Potassium	884	907	2.57
	Magnesium	1,510	1,550	2.61
	Manganese	12.4	13.9	11.41
	Sodium	6,690	7,200	7.34
	C16	60.0	70.0	15.38
	C17	200	200	0.00
	C18	200	200	0.00
	C20	60.0	70.0	15.38
	C25	10.0	20.0	66.67

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
MI049U	Lead	15.4	17.2	11.04
	Barium	18.5	20.2	8.79
	Cadmium	85.5	88.7	3.67
	Calcium	3,920	4,210	7.13
	Iron	333	432	25.88
	Potassium	1,160	1,210	4.22
	Magnesium	574	589	2.58
	Manganese	47.9	54.4	12.71
	Sodium	3,540	3,620	2.23
MI049	Barium	10.6	12.1	13.22
	Cadmium	77.7	85.9	10.02
	Calcium	2,730	3,350	20.39
	Potassium	800	1,460	58.41
	Magnesium	531	641	18.77
	Manganese	17.3	27.7	46.22
	Sodium	3,460	4,060	15.96
	B2EHP	30.0	30.9	2.96
	C15	20.0	20.0	0.00
	C16	50.0	50.0	0.00
	C17	80.0	80.0	0.00
	C18	70.0	70.0	0.00
	C19	60.0	70.0	15.38
	C20	40.0	40.0	0.00
	C21	10.0	10.0	0.00
	2Prol	40.0	40.0	0.00
	246TNT	1.07	1.12	4.57

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
MI060U	Lead	4.01	6.29	44.27
	Aluminum	773	848	9.25
	Barium	53.2	53.8	1.12
	Calcium	5,370	5,680	5.61
	Iron	301	310	2.95
	Potassium	130,000	170,000	26.67
	Manganese	28.4	32.1	12.23
	Sodium	26,900	31,600	16.07
MI060	Aluminum	295	325	9.68
	Barium	36.0	37.0	2.74
	Calcium	4,170	4,260	2.14
	Potassium	180,000	190,000	5.41
	Manganese	3.88	3.99	2.80
	Sodium	25,800	27,500	6.38
	C15	20.0	30.0	40.00
	C16	50.0	100	66.67
	C17	100	100	0.00
	C18	50	100	66.67
	C19	50.0	100	66.67
	C20	40.0	50.0	22.22

SITE ID	ANALYTE	SAMPLE CONCENTRATION	DUPLICATE CONCENTRATION	%RPD
MI071U	Lead	7.05	11.7	49.60
	Aluminum	3,140	4,250	30.04
	Barium	66.0	68.3	3.43
	Cadmium	13.3	15.2	13.33
	Calcium	24,000	27,000	11.76
	Chromium	8.35	8.8	5.25
	Iron	2,760	3,080	10.96
	Potassium	7,150	7,220	0.97
	Magnesium	2,200	2,300	4.44
	Manganese	53.3	55.1	3.32
	Sodium	4,420	4,500	1.79
	Vanadium	8.14	8.47	3.97
	Zinc	29.4	30.3	3.02
MI071	Lead	1.63	1.95	17.88
	Barium	44.4	45.4	2.23
	Cadmium	11.7	12.6	7.41
	Calcium	14,600	15,500	5.98
	Potassium	9,000	9,280	3.06
	Magnesium	2,030	2,260	10.72
	Manganese	33.6	35.5	5.50
	Sodium	5,170	5,190	0.39
	Zinc	27.2	30.5	11.44
	C17	5.0	10.0	66.67

SEDIMENT DUPLICATE ASSESSMENT

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/G	DUPLICATE CONCENTRATION UG/G	%RPD
RVER-1	Lead	5.3	6.0	12.39
	Aluminum	4,589	6,353	32.24
	Arsenic	2.1	2.5	17.39
	Barium	75.5	92.1	19.81
	Calcium	443	300	-38.49
	Iron	4,173	4,777	13.50
	Potassium	302	440	37.20
	Magnesium	620	720	14.93
	Manganese	457	542	17.02
	Silver	0.10	0.10	0.00
	Sodium	384	372	-3.17
	Vanadium	14.6	12.6	-14.71
	CCL3F	0.01	0.01	0.00
	DIACAL	7.46	3.81	-64.77
DTCH1-1	Lead	9.9	19.9	67.11
	Aluminum	8,400	9,600	13.33
	Arsenic	6.1	10.6	53.89
	Barium	120	134	11.02
	Calcium	901	1,010	11.41
	Iron	16,000	19,000	17.14
	Potassium	541	644	17.38
	Magnesium	1,170	1,210	3.36
	Manganese	1,300	2,050	44.78
	Sodium	414	449	8.11
	Vanadium	37.9	42.5	11.44
	Acetone	0.08	0.09	11.76
	CHCL3F	0.01	0.02	66.67

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/G	DUPLICATE CONCENTRATION UG/G	%RPD
DTCH7-3	Lead	22.0	25.0	23.11
	Aluminum	6,800	6,815	0.22
	Arsenic	6.61	13.3	67.20
	Barium	161	164	1.85
	Calcium	801	1,300	47.50
	Iron	11,915	13,000	8.71
	Potassium	451	486	7.47
	Magnesium	884	994	11.71
	Manganese	1,330	1,573	16.74
	Silver	0.062	0.098	45.00
	Sodium	303	431	34.88
	Vanadium	33.2	34.6	4.13
DTCH4-4	Lead	12.7	16.7	27.21
	Aluminum	2,010	4,042	67.15
	Arsenic	12.4	20.9	51.05
	Calcium	439	632	36.04
	Iron	9,419	15,743	50.27
	Potassium	198	277	33.26
	Magnesium	189	381	67.37
	Manganese	658	2,845	124.86
	Silver	0.20	0.30	40.00
	Sodium	351	355	1.13
	Vanadium	39.7	39.3	-1.01

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/G	DUPLICATE CONCENTRATION UG/G	%RPD
DTCH10-5	Lead	11.1	14.0	23.11
	Aluminum	9,200	11,000	17.82
	Arsenic	8.29	8.92	7.32
	Barium	95.9	103	7.14
	Calcium	941	947	0.64
	Iron	13,000	14,000	7.41
	Potassium	466	694	39.31
	Magnesium	1,580	1,740	9.64
	Manganese	758	787	3.75
	Sodium	383	390	1.81
	Vanadium	37.7	39.3	4.16
	12EPHC	0.231	0.233	0.86
	2CHEL1L	0.117	0.231	65.52
DTCH9-3	Lead	11.0	23.2	71.35
	Aluminum	3,072	3,471	12.20
	Arsenic	5.2	6.0	14.29
	Barium	53.8	60.5	11.72
	Calcium	332	448	29.74
	Iron	9,906	10,625	7.00
	Mercury	0.20	0.30	40.00
	Potassium	265	295	10.71
	Magnesium	442	563	24.08
	Manganese	611	673	9.66
	Sodium	333	386	14.74
	Vanadium	18.2	18.7	2.71
	Zinc	59.0	60.6	2.68
	12EPCH	0.00	0.11	200.00

SURFACE AND SUBSURFACE SOIL DUPLICATE ASSESSMENT

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
ADAB-1	Lead	1.4	1.5	6.90
	Chromium	58.5	71.6	20.14
ADAB-2	Lead	8.5	12.0	34.15
CBG-3	Lead	6.0	8.4	33.33
CBG-4	Lead	7.5	9.3	21.43
OBGA-2	Vanadium	11.4	15.0	27.27
	Lead	3.5	4.6	27.16
	Arsenic	2.7	3.5	25.81
	Aluminum	6100.0	6800.0	10.85
	Calcium	175.0	216.0	20.97
	Iron	5700.0	6500.0	13.11
	Potassium	221.0	287.0	25.98
	Magnesium	519.0	618.0	17.41
	Manganese	65.6	76.7	15.60
	Sodium	301.0	368.0	20.03
CDP-1A	Lead	3.9	4.6	16.47
CDP-1B	Vanadium	7.2	10.1	33.53
	Lead	1.5	2.7	57.14
	Arsenic	1.1	1.5	30.77
	Aluminum	1867.5	2601.7	32.86
	Calcium	211.9	231.5	8.84
	Iron	2526.5	3129.6	21.33
	Potassium	75.5	102.0	29.86
	Magnesium	134.9	178.4	27.77
	Manganese	36.2	41.0	12.44
	Sodium	303.1	372.2	20.47
	2PROL	0.022	0.032	37.04

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
OBGA-4	Lead	11.4	48.3	123.62
	Vanadium	21.5	25.7	17.80
	Arsenic	5.2	5.74.6	196.41
	Aluminum	9200.0	11000.0	17.82
	Barium	90.3	103.0	13.14
	Calcium	600.0	662.0	9.83
	Iron	11000.0	12000.0	8.70
	Potassium	295.0	397.0	29.48
	Magnesium	922.0	1160.0	22.86
	Manganese	125.0	127.0	1.59
	Sodium	447.0	459.0	2.65
	RDX	1.1	1.1	0.00
CLF-3	Vanadium	17.5	28.7	48.48
	Lead	6.3	8.8	33.11
	Arsenic	1.8	2.7	40.00
	Aluminum	2251.2	7377.0	106.47
	Barium	62.5	158.7	86.98
	Calcium	533.7	850.3	45.75
	Iron	4784.9	9615.4	67.09
	Potassium	151.6	351.1	79.37
	Magnesium	285.4	1368.6	130.98
	Manganese	93.8	408.6	125.32
	Sodium	386.0	636.6	49.01
	ANAPNE	1.13	0.11	-164.52
	2PROL	0.059	0.492	157.17
	ACET	0.623	8.29	172.04
OBGB-3	Lead	0.4	0.3	-28.57
OBGB-4	Lead	5.58	1.05	-136.65
OBGB-5	Lead	16.0	10.8	-38.81

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
OBGB-5	Mercury	0.1	0.1	0.00
OBGC-3	Lead	0.553	0.702	23.75
OBGA-4S	Lead	152.0	120.0	-23.53
	HMX	67.0	31.0	-73.47
LF-1	Lead	9.3	14.9	46.28
	Arsenic	5.1	5.3	3.85
	Aluminum	13064.1	14234.9	8.58
	Barium	122.2	128.2	4.79
	Calcium	613.4	641.1	4.42
	Iron	12561.7	13687.4	8.58
	Potassium	532.4	581.7	8.85
	Magnesium	1224.0	1614.1	27.49
	Manganese	247.9	316.0	24.15
	Sodium	261.5	336.2	25.00
	Vanadium	40.0	41.1	2.71
LF-2	Lead	9.3	19.4	70.38
SA-7	Lead	10.1	13.0	25.11
	Arsenic	3.37	3.73	10.14
	Aluminum	11288.3	12058.5	6.60
	Barium	186.1	214.9	14.36
	Calcium	1021.5	1027.9	0.62
	Iron	14054.2	14157.6	0.73
	Potassium	470.3	679.6	36.40
	Magnesium	1256.8	2788.6	75.73
	Manganese	1716.3	2580.2	40.21
	Sodium	314.2	346.5	9.78
	Lead	10.9	14.3	26.98
	Vanadium	38.3	41.6	8.26
	12CHE1L	0.245	0.365	39.34

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
SB-18	Lead	4.08	4.11	0.73
	246TNT	2.13	2.38	11.09
	HMX	0.847	0.906	6.73
	RDX	7.56	8.08	6.65
SC-5	Lead	6.1	7.0	13.74
SC-42E	Lead	10.0	10.3	2.96
	Arsenic	6.7	9.46	34.16
	Aluminum	16000.0	20000.0	22.22
	Barium	230.0	231.0	0.43
	Calcium	1240.0	1300.0	4.72
	Iron	21000.0	23000.0	9.09
	Potassium	617.0	989.0	46.33
	Magnesium	2150.0	2490.0	14.66
	Manganese	298.0	299.0	0.34
	Sodium	438.0	523.0	17.69
	Vanadium	40.9	42.9	4.77
	12EPCH	0.246	0.247	0.41
	246TNT	0.699	0.774	10.18
	RDX	0.659	0.663	0.61
SD-42	Lead	10.0	11.1	10.43
	Vanadium	22.3	35.1	44.60
	Arsenic	4.44	6.22	33.40
	Aluminum	5624.3	6404.5	12.97
	Barium	141.5	357.7	86.62
	Calcium	1350.1	1518.1	11.71
	Iron	8859.1	8869.1	0.11
	Potassium	319.2	403.1	23.23
	Magnesium	710.7	715.0	0.60
	Manganese	749.9	896.1	17.76

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
SD-42	Sodium	374.4	410.8	9.27
	NAP	0.118	0.144	19.85
	HMX	2.5	8.6	109.91
SO-14	Lead	10.4	10.6	1.90
	Arsenic	3.6	3.86	6.97
	Aluminum	7764.7	8009.4	3.10
	Barium	107.9	116.1	7.32
	Calcium	658.1	1015.1	42.67
	Iron	10067.9	10419.5	3.43
	Potassium	357.9	388.9	8.30
	Magnesium	991.1	1145.7	14.47
	Manganese	562.6	574.3	2.06
	Vanadium	33.1	35.2	6.15
	246TNT	0.725	0.763	5.11
SE-4	Lead	4.2	4.2	0.00
SZ-4W	Lead	11.7	12.0	2.53
	Arsenic	7.76	9.35	18.59
	Aluminum	20000.0	21000.0	4.88
	Barium	127.0	133.0	4.62
	Calcium	874.0	963.0	9.69
	Iron	21000.0	22000.0	4.65
	Potassium	790.0	860.0	8.48
	Magnesium	1370.0	1550.0	12.33
	Manganese	418.0	517.0	21.18
	Vanadium	49.8	54.6	9.20
	Sodium	705.0	780.0	10.10
	ACET	0.096	0.117	19.72
SZ-2	Lead	9.82	11.0	11.34
SX-26	Lead	12.5	12.1	-3.25

SITE ID	ANALYTE	SAMPLE CONCENTRATION UG/L	DUPLICATE CONCENTRATION UG/L	%RPD
SX-26	Arsenic	4.44	7.07	45.70
	Aluminum	13000.0	15000.0	14.29
	Barium	153.0	168.0	9.35
	Calcium	2290.0	2850.0	21.79
	Iron	17000.0	21000.0	21.05
	Potassium	594.0	686.0	14.38
	Magnesium	1900.0	2340.0	20.75
	Manganese	579.0	777.0	29.20
	Vanadium	35.3	37.3	5.51
	Zinc	82.1	231.0	95.11
SYD-1	Lead	14.0	20.6	38.15
	Arsenic	4.5	4.8	6.45
	Aluminum	9665.9	11418.3	16.62
	Barium	130.1	134.5	3.33
	Calcium	811.1	897.0	10.06
	Iron	10441.5	11557.0	10.14
	Potassium	345.1	500.1	36.68
	Magnesium	1710.4	2130.7	21.88
	Manganese	836.9	1126.4	29.49
	Sodium	363.3	465.0	24.56
	Vanadium	32.3	36.6	12.48

CONTAMINATION ASSOCIATED WITH FIELD BLANKS

CONTAMINATION ASSOCIATED WITH MAAP TRIP BLANKS

SAMPLE ID	SAMPLING DATE	ANALYTE	CONCENTRATION (ug/L)
GWTB-05	10/28/90	Carbon Disulfide	160
GWTB-14	11/04/90	Acetone	28
SETB-008	08/20/90	Acetone	18
SETB-010	08/22/90	Acetone	18
SETB-011	08/23/90	1,2-Dichloroethane	0.79
SETB-013	08/26/90	Carbon Disulfide	1.00
SETB-014	09/12/90	Acetone Carbon Disulfide	20 84
SETB-014	09/12/90	Trichlorofluoromethane	4.5
SETB-016	09/13/90	Carbon Disulfide	12
SOTB-15	11/09/90	Acetone	22
SOTB3	08/22/90	Chloroform	0.61

CONTAMINANTS DETECTED IN RINSE BLANKS COLLECTED AT MAAP

SAMPLE ID	SAMPLING DATE	ANALYTE	CONCENTRATION (ug/L)
SORB-15	10/05/90	Barium Calcium Copper Iron Magnesium Manganese Sodium	7.51 1550 13.1 228 583 3.2 2340
SORB-16	10/06/90	Barium Chromium Iron	5.95 7.36 86.5
SORB-17	10/10/90	Barium Calcium Copper Iron Manganese	10.2 2820 12.8 561 9.5
GWRB-001	10/29/90	Calcium Iron Sodium	579 65.0 614
GWRB-002	11/01/90	Iron	46.7
GWRB-003	11/02/90	Barium	5.39
GWRB-004	11/03/90	Copper	11.1
SORB1	07/27/90	Zinc Lead	30.7 1.5
SERB-004	08/20/90	Zinc Lead	24.6 2.7
SORB-4	08/24/90	Lead	2.6
SERB-006	08/26/90	Lead	2.2
SERB-007	08/26/90	Lead	14.3
SORB-7	09/10/90	Lead	1.8
SERB-009	09/13/90	Lead	2.1
SORB-12	09/20/90	Zinc	29.9
SORB-15	10/05/90	Zinc	26.8
SORB-17	10/10/90	Zinc Lead	38.2 4.0

CONTAMINANTS DETECTED IN RINSE BLANKS COLLECTED AT MAAP

SAMPLE ID	SAMPLING DATE	ANALYTE	CONCENTRATION (ug/L)
SORB1	07/27/90	Manganese Sodium	4.7 862
SERB-001	08/06/90	Copper	20.3
SERB-002	08/10/90	Copper Manganes	14.9 3.6
SORB2	08/13/90	Copper Manganese	39.2 3.2
SERB-003	08/15/90	Manganese	3.6
SERB-004	08/20/90	Barium Calcium Copper Iron Magnesium Manganese Sodium	9.15 2130 8.32 282 659 38.0 2870
SORB3	08/22/90	Manganese	4.8
SORB-4	08/24/90	Manganese	3.6
SERB-006	08/26/90	Manganese	3.5
SERB-007	08/26/90	Manganese	6.9
SORB-7	09/10/90	Iron	67.2
SERB-009	09/13/90	Barium	10.3
SORB-12	09/20/90	Barium Iron Magnesium Sodium	9.92 249 640 2410
SORB-13	09/24/90	Copper Iron	19.0 87.8
SORB-14	09/26/90	Calcium Copper Iron Manganese	852 12.1 95.4 3.9

APPENDIX N

LOGBOOK FOR GROUNDWATER FLOW MODEL

```
*****************************************************************
*                       E C H O P R I N T                       *
*                                                               *
*                       F L O W P A T H                         *
*                         version 3.0                           *
*                                                               *
*  FLOWPATH was written by Thomas Franz and Nilson Guiguer      *
*                                                               *
*****************************************************************
*                                                               *
*                    Copyright 1989, 1990                       *
*                            by                                 *
*                 Waterloo Hydrogeologic Software               *
*                      113-106 Seagram Drive                    *
*                        Waterloo, Ontario                      *
*                         N2L 3B8, Canada                       *
*                                                               *
*                       ph (519) 746-1798                       *
*                                                               *
*****************************************************************
```

FLOWPATH logbook for data set : MAAPMOD

Unit System : English units [ft/gal/d]

***** GRID PARAMETERS *****

Number of x-grid lines : 53

Number of y-grid lines : 56

Grid coordinates (x-grid lines) [ft] :

1	3.41000E+02
2	3.41133E+02
3	3.41426E+02
4	3.41693E+02
5	3.41959E+02
6	3.42226E+02
7	3.42492E+02
8	3.42759E+02
9	3.43025E+02
10	3.43292E+02
11	3.43558E+02

12	3.43825E+02
13	3.44091E+02
14	3.44384E+02
15	3.44651E+02
16	3.44917E+02
17	3.45184E+02
18	3.45450E+02
19	3.45717E+02
20	3.45983E+02
21	3.46250E+02
22	3.46516E+02
23	3.46783E+02
24	3.47049E+02
25	3.47316E+02
26	3.47582E+02
27	3.47742E+02
28	3.47876E+02
29	3.48009E+02
30	3.48142E+02
31	3.48275E+02
32	3.48435E+02
33	3.48542E+02
34	3.48702E+02
35	3.48808E+02
36	3.48968E+02
37	3.49075E+02
38	3.49235E+02
39	3.49368E+02
40	3.49501E+02
41	3.49768E+02
42	3.50034E+02
43	3.50301E+02
44	3.50567E+02
45	3.50834E+02
46	3.51127E+02
47	3.51393E+02
48	3.51660E+02
49	3.51926E+02
50	3.52193E+02
51	3.52459E+02
52	3.52726E+02
53	3.53000E+02

Grid coordinates (y-grid lines) [ft] :

1	3.96900E+03
2	3.96926E+03
3	3.96951E+03
4	3.96977E+03
5	3.97002E+03
6	3.97022E+03
7	3.97047E+03
8	3.97073E+03

9	3.97098E+03
10	3.97111E+03
11	3.97124E+03
12	3.97137E+03
13	3.97149E+03
14	3.97162E+03
15	3.97175E+03
16	3.97188E+03
17	3.97201E+03
18	3.97213E+03
19	3.97226E+03
20	3.97245E+03
21	3.97271E+03
22	3.97297E+03
23	3.97322E+03
24	3.97348E+03
25	3.97367E+03
26	3.97392E+03
27	3.97405E+03
28	3.97418E+03
29	3.97431E+03
30	3.97444E+03
31	3.97456E+03
32	3.97469E+03
33	3.97482E+03
34	3.97495E+03
35	3.97508E+03
36	3.97520E+03
37	3.97540E+03
38	3.97565E+03
39	3.97591E+03
40	3.97616E+03
41	3.97642E+03
42	3.97667E+03
43	3.97693E+03
44	3.97712E+03
45	3.97738E+03
46	3.97763E+03
47	3.97789E+03
48	3.97815E+03
49	3.97840E+03
50	3.97866E+03
51	3.97885E+03
52	3.97910E+03
53	3.97936E+03
54	3.97962E+03
55	3.97987E+03
56	3.98000E+03

***** WELL PARAMETERS *****

Number of wells : 2

No.	i	j	X [ft]	Y [ft]	well discharge [gpd]
1	41	29	3.49768E+02	3.97431E+03	-3.26000E-07
2	15	42	3.44651E+02	3.97667E+03	-1.25000E-06

***** CONSTRAINED HEAD NODES *****

Number of constant head nodes : 129

No.	i	j	X [ft]	Y [ft]	const. head [ft]
1	7	22	3.42492E+02	3.97297E+03	1.18567E-01
2	7	21	3.42492E+02	3.97271E+03	1.18872E-01
3	7	23	3.42492E+02	3.97322E+03	1.18262E-01
4	7	26	3.42492E+02	3.97392E+03	1.17348E-01
5	7	37	3.42492E+02	3.97540E+03	1.16052E-01
6	7	31	3.42492E+02	3.97456E+03	1.16738E-01
7	7	24	3.42492E+02	3.97348E+03	1.17958E-01
8	7	33	3.42492E+02	3.97482E+03	1.16434E-01
9	7	36	3.42492E+02	3.97520E+03	1.15976E-01
10	45	37	3.50834E+02	3.97533E+03	1.17120E-01
11	7	39	3.42492E+02	3.97591E+03	1.15824E-01
12	7	27	3.42492E+02	3.97405E+03	1.17272E-01
13	7	28	3.42492E+02	3.97418E+03	1.17196E-01
14	7	25	3.42492E+02	3.97367E+03	1.17653E-01
15	7	30	3.42492E+02	3.97444E+03	1.16891E-01
16	7	29	3.42492E+02	3.97431E+03	1.17043E-01
17	7	34	3.42492E+02	3.97495E+03	1.16281E-01
18	7	35	3.42492E+02	3.97508E+03	1.16129E-01
19	7	38	3.42492E+02	3.97565E+03	1.16022E-01
20	40	45	3.49501E+02	3.97738E+03	1.12441E-01
21	37	46	3.49075E+02	3.97763E+03	1.12150E-01
22	42	44	3.50034E+02	3.97712E+03	1.12776E-01
23	43	44	3.50301E+02	3.97712E+03	1.13599E-01
24	44	44	3.50567E+02	3.97712E+03	1.14422E-01
25	45	44	3.50834E+02	3.97712E+03	1.14910E-01
26	26	48	3.47582E+02	3.97815E+03	1.11069E-01
27	24	50	3.47049E+02	3.97866E+03	1.10734E-01
28	23	50	3.46783E+02	3.97866E+03	1.10399E-01
29	22	51	3.46516E+02	3.97885E+03	1.09728E-01
30	19	52	3.45717E+02	3.97910E+03	1.09728E-01
31	41	44	3.49768E+02	3.97712E+03	1.12608E-01
32	39	45	3.49368E+02	3.97738E+03	1.12329E-01
33	38	45	3.49235E+02	3.97738E+03	1.12217E-01
34	33	47	3.48542E+02	3.97789E+03	1.11770E-01
35	31	47	3.48275E+02	3.97789E+03	1.11526E-01
36	32	47	3.48435E+02	3.97789E+03	1.11648E-01

37	30	48	3.48142E+02	3.97815E+03	1.11404E-01
38	30	47	3.48142E+02	3.97789E+03	1.11404E-01
39	40	44	3.49501E+02	3.97712E+03	1.12608E-01
40	33	46	3.48542E+02	3.97763E+03	1.11770E-01
41	36	46	3.48968E+02	3.97763E+03	1.12022E-01
42	35	46	3.48808E+02	3.97763E+03	1.11938E-01
43	34	46	3.48702E+02	3.97763E+03	1.11854E-01
44	29	48	3.48009E+02	3.97815E+03	1.11320E-01
45	28	48	3.47876E+02	3.97815E+03	1.11237E-01
46	27	48	3.47742E+02	3.97815E+03	1.11153E-01
47	24	49	3.47049E+02	3.97840E+03	1.10734E-01
48	26	49	3.47582E+02	3.97840E+03	1.11069E-01
49	25	49	3.47316E+02	3.97840E+03	1.10901E-01
50	19	51	3.45717E+02	3.97885E+03	1.09728E-01
51	20	51	3.45983E+02	3.97885E+03	1.09728E-01
52	21	51	3.46250E+02	3.97885E+03	1.09728E-01
53	14	51	3.44384E+02	3.97885E+03	1.12776E-01
54	9	45	3.43025E+02	3.97738E+03	1.15062E-01
55	10	47	3.43292E+02	3.97789E+03	1.14300E-01
56	12	49	3.43825E+02	3.97840E+03	1.13538E-01
57	13	51	3.44091E+02	3.97885E+03	1.12776E-01
58	13	50	3.44091E+02	3.97866E+03	1.13157E-01
59	11	49	3.43558E+02	3.97840E+03	1.13538E-01
60	10	48	3.43292E+02	3.97815E+03	1.14300E-01
61	11	48	3.43558E+02	3.97815E+03	1.13919E-01
62	9	47	3.43025E+02	3.97789E+03	1.14300E-01
63	9	46	3.43025E+02	3.97763E+03	1.14681E-01
64	12	50	3.43825E+02	3.97866E+03	1.13538E-01
65	15	53	3.44651E+02	3.97936E+03	1.12166E-01
66	15	52	3.44651E+02	3.97910E+03	1.12471E-01
67	14	52	3.44384E+02	3.97910E+03	1.12471E-01
68	15	54	3.44651E+02	3.97962E+03	1.11557E-01
69	15	55	3.44651E+02	3.97987E+03	1.09728E-01
70	16	55	3.44917E+02	3.97987E+03	1.09728E-01
71	17	54	3.45184E+02	3.97962E+03	1.09728E-01
72	18	53	3.45450E+02	3.97936E+03	1.09728E-01
73	18	52	3.45450E+02	3.97910E+03	1.09728E-01
74	17	53	3.45184E+02	3.97936E+03	1.09728E-01
75	16	54	3.44917E+02	3.97962E+03	1.10642E-01
76	8	44	3.42759E+02	3.97712E+03	1.15824E-01
77	8	43	3.42759E+02	3.97693E+03	1.15824E-01
78	8	42	3.42759E+02	3.97667E+03	1.15824E-01
79	8	41	3.42759E+02	3.97642E+03	1.15824E-01
80	8	40	3.42759E+02	3.97616E+03	1.15824E-01
81	8	39	3.42759E+02	3.97591E+03	1.15824E-01
82	45	36	3.50834E+02	3.97520E+03	1.18872E-01
83	43	42	3.50301E+02	3.97667E+03	1.13538E-01
84	44	41	3.50567E+02	3.97642E+03	1.15214E-01
85	45	38	3.50834E+02	3.97565E+03	1.15824E-01
86	45	40	3.50834E+02	3.97616E+03	1.15062E-01
87	44	40	3.50567E+02	3.97616E+03	1.15062E-01
88	45	39	3.50834E+02	3.97591E+03	1.15443E-01
89	44	42	3.50567E+02	3.97667E+03	1.13538E-01
90	43	43	3.50301E+02	3.97693E+03	1.13568E-01

91	39	2	3.49368E+02	3.96926E+03	1.26492E-01
92	26	2	3.47582E+02	3.96926E+03	1.24968E-01
93	18	2	3.45450E+02	3.96926E+03	1.23444E-01
94	14	2	3.44384E+02	3.96926E+03	1.21920E-01
95	13	2	3.44091E+02	3.96926E+03	1.21920E-01
96	12	2	3.43825E+02	3.96926E+03	1.21920E-01
97	11	2	3.43558E+02	3.96926E+03	1.21920E-01
98	10	2	3.43292E+02	3.96926E+03	1.21920E-01
99	9	2	3.43025E+02	3.96926E+03	1.21920E-01
100	8	2	3.42759E+02	3.96926E+03	1.21920E-01
101	7	2	3.42492E+02	3.96926E+03	1.21920E-01
102	15	2	3.44651E+02	3.96926E+03	1.22301E-01
103	16	2	3.44917E+02	3.96926E+03	1.22682E-01
104	17	2	3.45184E+02	3.96926E+03	1.23063E-01
105	19	2	3.45717E+02	3.96926E+03	1.23635E-01
106	20	2	3.45983E+02	3.96926E+03	1.23825E-01
107	21	2	3.46250E+02	3.96926E+03	1.24016E-01
108	22	2	3.46516E+02	3.96926E+03	1.24206E-01
109	23	2	3.46783E+02	3.96926E+03	1.24397E-01
110	24	2	3.47049E+02	3.96926E+03	1.24588E-01
111	25	2	3.47316E+02	3.96926E+03	1.24778E-01
112	27	2	3.47742E+02	3.96926E+03	1.25085E-01
113	28	2	3.47876E+02	3.96926E+03	1.25202E-01
114	29	2	3.48009E+02	3.96926E+03	1.25320E-01
115	30	2	3.48142E+02	3.96926E+03	1.25437E-01
116	31	2	3.48275E+02	3.96926E+03	1.25554E-01
117	32	2	3.48435E+02	3.96926E+03	1.25671E-01
118	33	2	3.48542E+02	3.96926E+03	1.25788E-01
119	34	2	3.48702E+02	3.96926E+03	1.25906E-01
120	35	2	3.48808E+02	3.96926E+03	1.26023E-01
121	36	2	3.48968E+02	3.96926E+03	1.26140E-01
122	37	2	3.49075E+02	3.96926E+03	1.26257E-01
123	38	2	3.49235E+02	3.96926E+03	1.26374E-01
124	40	2	3.49501E+02	3.96926E+03	1.26492E-01
125	41	2	3.49768E+02	3.96926E+03	1.26492E-01
126	42	2	3.50034E+02	3.96926E+03	1.26644E-01
127	43	2	3.50301E+02	3.96926E+03	1.26797E-01
128	44	2	3.50567E+02	3.96926E+03	1.26949E-01
129	45	2	3.50834E+02	3.96926E+03	1.27102E-01

***** SPECIFIED FLUX NODES *****

Number of flux nodes : 0

***** SURFACE WATER BODIES *****

Number of surface water body nodes : 0

***** AQUIFER PROPERTIES *****

Number of different material properties : 1

No.	Kxx [ft/d]	Kyy [ft/d]	Porosity [-]	
1	8.20000E-02	8.20000E-03	2.00000E-01	(default)

********** DISTRIBUTION OF AQUIFER MATERIAL PROPERTIES **********

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1
52	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1	1
51	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1	1	1
50	*	*	*	*	*	*	*	*	*	*	*	1	1	1	1	1	1
49	*	*	*	*	*	*	*	*	*	*	1	1	1	1	1	1	1
48	*	*	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1
47	*	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1
46	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
45	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
44	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
43	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
42	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
41	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
40	*	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1
39	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
38	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
37	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
36	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
35	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
34	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
33	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
32	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
31	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
30	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
29	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
28	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
27	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
26	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
25	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
24	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
23	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
22	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
21	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1

20	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
19	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
18	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
17	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
16	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
15	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
14	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
13	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
12	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
11	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
10	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
9	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
8	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
7	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
6	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
5	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
4	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
3	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
2	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
1	*	*	*	*	*	*	1	1	1	1	1	1	1	1	1	1	1
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	1	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	1	1	1	1	*	*	*	*	*	*	*	*	*	*	*	*	*
51	1	1	1	1	1	1	*	*	*	*	*	*	*	*	*	*	*
50	1	1	1	1	1	1	1	1	*	*	*	*	*	*	*	*	*
49	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*	*
48	1	1	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*
47	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	*
46	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
45	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
44	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
43	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
42	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
41	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
40	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
39	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
38	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
37	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
36	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
35	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
34	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
33	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
32	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
31	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
30	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
29	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

28	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
27	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
26	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
25	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
24	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
23	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
22	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
21	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
19	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
18	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
17	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
16	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
15	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
14	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
13	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
12	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
11	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
10	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
9	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
8	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
7	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
6	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
5	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34

	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
51	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
50	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
49	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
48	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
47	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
46	1	1	1	*	*	*	*	*	*	*	*	*	*	*	*	*	*
45	1	1	1	1	1	1	*	*	*	*	*	*	*	*	*	*	*
44	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
43	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
42	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
41	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
40	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
39	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
38	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
37	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*

	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51
36	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
35	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
34	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
33	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
32	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
31	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
30	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
29	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
28	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
27	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
26	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
25	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
24	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
23	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
22	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
21	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
20	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
19	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
18	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
17	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
16	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
15	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
14	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
13	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
12	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
11	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
10	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
9	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
8	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
7	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
6	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
5	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
4	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
3	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
2	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
1	1	1	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*

	52	53
56	*	*
55	*	*
54	*	*
53	*	*
52	*	*
51	*	*
50	*	*
49	*	*
48	*	*
47	*	*
46	*	*
45	*	*

44	*	*
43	*	*
42	*	*
41	*	*
40	*	*
39	*	*
38	*	*
37	*	*
36	*	*
35	*	*
34	*	*
33	*	*
32	*	*
31	*	*
30	*	*
29	*	*
28	*	*
27	*	*
26	*	*
25	*	*
24	*	*
23	*	*
22	*	*
21	*	*
20	*	*
19	*	*
18	*	*
17	*	*
16	*	*
15	*	*
14	*	*
13	*	*
12	*	*
11	*	*
10	*	*
9	*	*
8	*	*
7	*	*
6	*	*
5	*	*
4	*	*
3	*	*
2	*	*
1	*	*
	52	53

***** AQUIFER TYPE *****

Unconfined aquifer

***** AQUIFER BOTTOM ELEVATIONS *****

Number of different aquifer bottom elevations : 9

No.	aquifer bottom elevation [ft]
1	3.05000E-02 (default)
2	3.05000E-02
3	3.81250E-02
4	4.57500E-02
5	5.33750E-02
6	6.10000E-02
7	6.86250E-02
8	7.62500E-02
9	9.15000E-02

********** DISTRIBUTION OF AQUIFER BOTTOM ELEVATIONS **********

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	2
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	2
52	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1	2
51	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1	1	2
50	*	*	*	*	*	*	*	*	*	*	*	1	1	1	2	2	2
49	*	*	*	*	*	*	*	*	*	*	1	1	1	1	2	2	2
48	*	*	*	*	*	*	*	*	*	1	1	1	1	1	2	2	2
47	*	*	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2
46	*	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2
45	*	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2
44	*	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2
43	*	*	*	*	*	*	*	1	1	1	1	2	2	2	2	2	2
42	*	*	*	*	*	*	*	1	1	1	1	2	2	2	2	2	2
41	*	*	*	*	*	*	*	1	1	1	1	2	2	2	2	3	3
40	*	*	*	*	*	*	*	1	1	1	1	2	2	2	2	3	3
39	*	*	*	*	*	*	1	1	1	1	1	2	2	2	2	3	3
38	*	*	*	*	*	*	1	1	1	2	2	2	2	2	2	3	3
37	*	*	*	*	*	*	1	1	1	2	2	2	2	2	2	3	3
36	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3
35	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3
34	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3
33	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3
32	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3
31	*	*	*	*	*	*	1	1	1	2	2	2	2	3	3	3	3

30	*	*	*	*	*	*	1	1	2	2	2	2	2	3	3	3	3
29	*	*	*	*	*	*	1	1	2	2	2	2	2	3	3	3	3
28	*	*	*	*	*	*	1	1	2	2	2	3	3	3	3	3	3
27	*	*	*	*	*	*	1	1	2	2	2	3	3	3	3	3	3
26	*	*	*	*	*	*	1	1	2	2	2	3	3	3	3	3	3
25	*	*	*	*	*	*	1	1	2	2	2	3	3	3	3	3	3
24	*	*	*	*	*	*	1	1	1	2	2	3	3	3	3	4	4
23	*	*	*	*	*	*	1	1	1	2	2	3	3	3	3	4	4
22	*	*	*	*	*	*	1	1	1	3	3	3	3	3	3	4	4
21	*	*	*	*	*	*	1	1	1	3	3	3	3	3	3	4	4
20	*	*	*	*	*	*	1	1	1	3	3	3	3	3	3	4	4
19	*	*	*	*	*	*	1	1	1	3	3	3	3	4	4	4	4
18	*	*	*	*	*	*	1	1	1	3	3	3	3	4	4	4	4
17	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
16	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
15	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
14	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
13	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
12	*	*	*	*	*	*	1	3	3	3	3	3	3	4	4	4	4
11	*	*	*	*	*	*	3	3	3	3	3	3	3	4	4	4	4
10	*	*	*	*	*	*	3	3	3	3	3	4	4	4	4	4	4
9	*	*	*	*	*	*	3	3	3	3	3	4	4	4	4	4	4
8	*	*	*	*	*	*	3	3	3	3	3	4	4	4	4	4	4
7	*	*	*	*	*	*	3	3	3	4	4	4	4	4	4	4	4
6	*	*	*	*	*	*	3	3	3	4	4	4	4	4	4	5	5
5	*	*	*	*	*	*	3	4	4	4	4	4	4	4	4	5	5
4	*	*	*	*	*	*	3	4	4	4	4	4	4	5	5	5	5
3	*	*	*	*	*	*	3	4	4	4	4	4	4	5	5	5	5
2	*	*	*	*	*	*	3	4	4	4	4	4	4	5	5	5	5
1	*	*	*	*	*	*	3	4	4	4	4	4	4	5	5	5	5

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34

56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	2	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	2	2	2	3	*	*	*	*	*	*	*	*	*	*	*	*	*
51	2	2	2	3	3	3	*	*	*	*	*	*	*	*	*	*	*
50	2	2	3	3	3	3	4	4	*	*	*	*	*	*	*	*	*
49	2	2	3	3	3	3	4	4	4	4	*	*	*	*	*	*	*
48	2	2	3	3	3	3	4	4	4	4	5	5	5	*	*	*	*
47	2	2	3	3	3	3	4	4	4	4	5	5	5	5	5	5	*
46	3	3	3	3	4	4	4	4	4	4	5	5	5	5	5	5	5
45	3	3	3	3	4	4	4	4	4	4	5	5	5	5	5	5	5
44	3	3	3	3	4	4	4	4	4	4	5	5	5	5	5	5	5
43	3	3	3	3	4	4	4	4	4	4	5	5	5	5	5	5	5
42	3	3	3	3	4	4	4	4	4	4	5	5	5	5	5	5	5
41	3	3	3	4	4	4	5	5	5	5	5	5	5	5	6	6	6
40	3	3	3	4	4	4	5	5	5	5	5	5	5	5	6	6	6
39	3	3	3	4	4	4	5	5	5	5	5	5	5	5	6	6	6
38	3	3	4	4	4	4	5	5	5	5	5	5	5	5	6	6	6

37	3	3	4	4	4	4	5	5	5	5	5	5	5	5	6	6	6
36	3	3	4	4	4	4	5	5	5	5	5	5	5	5	6	6	6
35	3	3	4	4	4	4	5	5	5	5	6	6	6	6	6	6	6
34	3	3	4	4	4	4	5	5	5	5	6	6	6	6	6	6	6
33	4	4	4	4	4	4	5	5	5	5	6	6	6	6	6	6	6
32	4	4	4	4	4	4	5	5	5	5	6	6	6	6	6	6	6
31	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
30	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
29	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
28	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
27	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
26	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
25	4	4	4	4	5	5	5	5	5	5	6	6	6	6	6	6	6
24	4	4	4	4	5	5	5	6	6	6	6	6	6	6	6	6	6
23	4	4	4	5	5	5	5	6	6	6	6	6	6	6	6	6	6
22	4	4	4	5	5	5	5	6	6	6	6	6	6	6	7	7	7
21	4	4	4	5	5	5	5	6	6	6	6	6	6	6	7	7	7
20	4	4	5	5	5	5	5	6	6	6	6	6	6	6	7	7	7
19	4	4	5	5	5	5	6	6	6	6	6	6	6	6	7	7	7
18	4	4	5	5	5	5	6	6	6	6	6	6	6	6	7	7	7
17	4	4	5	5	5	5	6	6	6	6	6	6	6	6	7	7	7
16	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
15	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
14	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
13	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
12	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
11	5	5	5	5	5	5	6	6	6	6	7	7	7	7	7	7	7
10	5	5	5	5	6	6	6	6	6	6	7	7	7	7	7	7	7
9	5	5	5	5	6	6	6	6	6	6	7	7	7	7	7	7	7
8	5	5	5	5	6	6	6	6	6	6	7	7	7	7	7	7	7
7	5	5	5	5	6	6	6	6	6	6	7	7	7	7	7	7	7
6	5	5	5	5	6	6	6	6	6	6	7	7	7	7	7	7	7
5	5	5	5	5	6	6	6	7	7	7	7	7	7	7	7	7	7
4	5	5	5	5	6	6	6	7	7	7	7	7	7	7	7	7	7
3	5	5	5	5	6	6	6	7	7	7	7	7	7	7	8	8	8
2	5	5	5	5	6	6	6	7	7	7	7	7	7	7	8	8	8
1	5	5	5	5	6	6	6	7	7	7	7	7	7	7	8	8	8

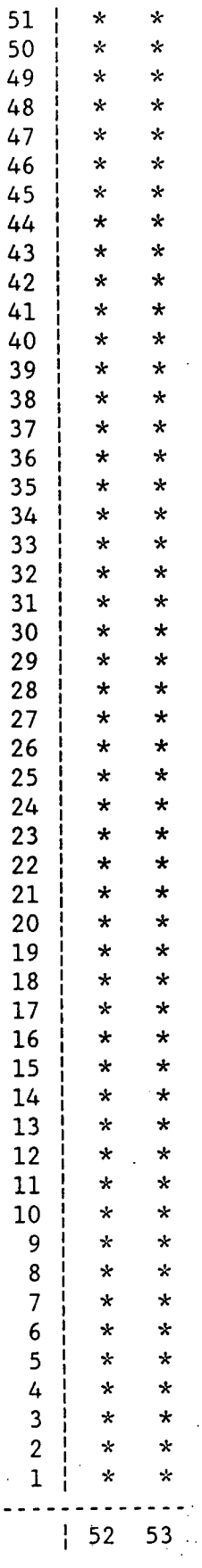
	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34
	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51

56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
51	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
50	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
49	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
48	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
47	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
46	5	6	6	*	*	*	*	*	*	*	*	*	*	*	*	*	*
45	5	6	6	6	6	6	*	*	*	*	*	*	*	*	*	*	*

44	5	6	6	6	6	6	6	7	7	7	7	*	*	*	*	*	*
43	5	6	6	6	6	6	6	7	7	7	7	*	*	*	*	*	*
42	5	6	6	6	6	6	6	7	7	7	7	*	*	*	*	*	*
41	6	6	6	6	6	6	6	7	7	7	7	*	*	*	*	*	*
40	6	6	6	6	6	6	6	7	7	7	7	*	*	*	*	*	*
39	6	6	6	6	6	7	7	7	7	7	7	*	*	*	*	*	*
38	6	6	6	6	6	7	7	7	7	7	7	*	*	*	*	*	*
37	6	6	6	6	6	7	7	7	7	7	7	*	*	*	*	*	*
36	6	6	6	6	6	7	7	7	7	8	8	*	*	*	*	*	*
35	6	6	6	6	6	7	7	7	7	8	8	*	*	*	*	*	*
34	6	6	6	6	6	7	7	7	7	8	8	*	*	*	*	*	*
33	6	6	6	6	6	7	7	7	7	8	8	*	*	*	*	*	*
32	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
31	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
30	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
29	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
28	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
27	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
26	6	7	7	7	7	7	7	7	7	8	8	*	*	*	*	*	*
25	6	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
24	6	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
23	6	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
22	7	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
21	7	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
20	7	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
19	7	7	7	7	7	7	7	8	8	8	8	*	*	*	*	*	*
18	7	7	7	7	7	8	8	8	8	8	8	*	*	*	*	*	*
17	7	7	7	7	7	8	8	8	8	8	8	*	*	*	*	*	*
16	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
15	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
14	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
13	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
12	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
11	7	7	7	7	7	8	8	8	8	9	9	*	*	*	*	*	*
10	7	8	8	8	8	8	8	8	8	9	9	*	*	*	*	*	*
9	7	8	8	8	8	8	8	8	8	9	9	*	*	*	*	*	*
8	7	8	8	8	8	8	8	8	8	9	9	*	*	*	*	*	*
7	7	8	8	8	8	8	8	8	8	9	9	*	*	*	*	*	*
6	7	8	8	8	8	8	8	8	8	9	9	*	*	*	*	*	*
5	7	8	8	8	8	8	8	9	9	9	9	*	*	*	*	*	*
4	7	8	8	8	8	8	8	9	9	9	9	*	*	*	*	*	*
3	8	8	8	8	8	8	8	9	9	9	9	*	*	*	*	*	*
2	8	8	8	8	8	8	8	9	9	9	9	*	*	*	*	*	*
1	8	8	8	8	8	8	8	9	9	9	9	*	*	*	*	*	*

	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51

	52	53
56	*	*
55	*	*
54	*	*
53	*	*
52	*	*



***** AREAL RECHARGE *****

Number of different infiltration/evapotranspiration rates : 2

No.	infiltration [L/T]	evapotranspiration [L/T]	effective recharge [L/T]	
1	6.40000E-07	0.00000E+00	6.40000E-07	(default)
1	1.60000E-07	0.00000E+00	1.60000E-07	

********** DISTRIBUTION OF AREAL IN/OUT-FLUXES **********

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	1	1
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	1	2	1
52	*	*	*	*	*	*	*	*	*	*	*	*	*	1	2	2	1
51	*	*	*	*	*	*	*	*	*	*	*	*	1	1	2	2	1
50	*	*	*	*	*	*	*	*	*	*	*	1	1	2	2	1	2
49	*	*	*	*	*	*	*	*	*	*	1	1	1	2	2	1	2
48	*	*	*	*	*	*	*	*	*	1	1	1	1	2	1	2	2
47	*	*	*	*	*	*	*	*	1	1	2	2	2	2	1	2	2
46	*	*	*	*	*	*	*	1	1	1	2	2	2	2	1	2	2
45	*	*	*	*	*	*	*	1	1	1	2	2	2	2	1	1	2
44	*	*	*	*	*	*	*	1	1	1	1	1	2	2	2	2	1
43	*	*	*	*	*	*	*	1	2	2	2	1	2	2	2	2	1
42	*	*	*	*	*	*	*	1	2	2	2	1	2	2	2	2	1
41	*	*	*	*	*	*	*	1	2	2	1	2	2	2	2	2	1
40	*	*	*	*	*	*	*	1	2	2	2	2	2	2	2	2	1
39	*	*	*	*	*	*	1	1	2	2	2	2	1	1	1	1	1
38	*	*	*	*	*	*	1	1	2	2	2	2	1	1	1	2	1
37	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	1
36	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	1
35	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	1
34	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2	2
33	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2	2
32	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2	2
31	*	*	*	*	*	*	1	1	1	1	1	1	2	2	2	2	2
30	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	2
29	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	2
28	*	*	*	*	*	*	1	1	2	2	2	1	2	2	2	2	2
27	*	*	*	*	*	*	1	1	2	2	2	1	1	1	1	1	2
26	*	*	*	*	*	*	1	1	2	1	1	1	1	1	1	1	2
25	*	*	*	*	*	*	1	1	2	1	1	1	2	2	2	2	2
24	*	*	*	*	*	*	1	1	2	1	1	1	2	2	2	2	2
23	*	*	*	*	*	*	1	1	2	1	1	1	1	1	2	1	2
22	*	*	*	*	*	*	1	2	2	1	1	1	1	1	2	1	1

21	*	*	*	*	*	*	1	2	1	1	2	2	2	1	1	1	1
20	*	*	*	*	*	*	1	2	1	1	2	2	2	1	1	1	1
19	*	*	*	*	*	*	2	1	1	1	2	2	2	2	2	2	2
18	*	*	*	*	*	*	2	1	1	1	1	1	1	2	2	2	2
17	*	*	*	*	*	*	2	1	1	1	1	1	1	2	2	2	2
16	*	*	*	*	*	*	2	1	1	1	1	1	1	1	2	2	2
15	*	*	*	*	*	*	2	2	2	2	1	1	2	2	2	2	2
14	*	*	*	*	*	*	2	2	2	2	1	1	2	2	2	2	2
13	*	*	*	*	*	*	2	2	2	2	1	1	1	1	2	2	2
12	*	*	*	*	*	*	2	2	2	2	1	1	1	1	2	2	2
11	*	*	*	*	*	*	2	2	2	2	2	2	1	1	1	1	1
10	*	*	*	*	*	*	2	2	2	2	2	2	1	1	1	1	1
9	*	*	*	*	*	*	2	2	2	2	2	2	1	2	1	1	2
8	*	*	*	*	*	*	2	2	2	2	2	2	1	2	2	1	1
7	*	*	*	*	*	*	2	2	2	2	2	2	1	2	2	1	2
6	*	*	*	*	*	*	2	2	2	2	2	2	1	2	2	1	2
5	*	*	*	*	*	*	2	2	2	2	1	1	1	2	1	1	1
4	*	*	*	*	*	*	2	2	2	2	1	1	1	1	2	2	2
3	*	*	*	*	*	*	2	1	1	1	1	1	1	1	1	1	1
2	*	*	*	*	*	*	2	1	1	2	1	2	1	2	2	1	1
1	*	*	*	*	*	*	2	1	1	1	1	1	1	1	1	1	1

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17

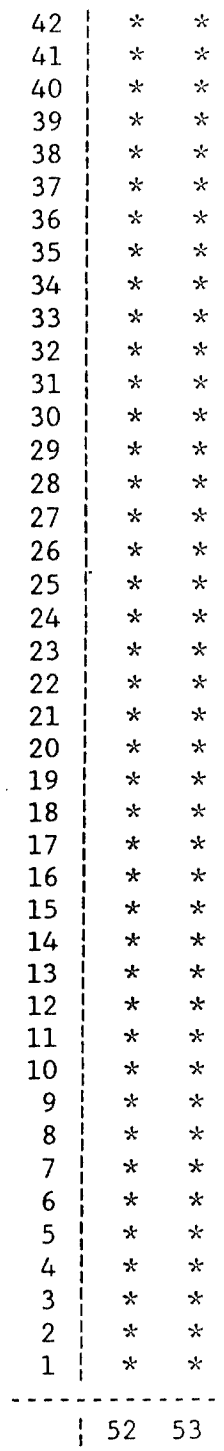
	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	1	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	1	1	1	1	*	*	*	*	*	*	*	*	*	*	*	*	*
51	2	2	2	1	1	1	*	*	*	*	*	*	*	*	*	*	*
50	2	2	2	1	1	1	1	1	*	*	*	*	*	*	*	*	*
49	2	2	2	1	1	2	2	1	1	1	*	*	*	*	*	*	*
48	2	2	2	1	1	2	2	1	1	1	1	1	1	*	*	*	*
47	2	2	2	1	2	2	2	1	2	2	1	1	1	1	1	1	*
46	2	2	2	1	2	2	2	1	2	2	1	1	1	1	1	1	1
45	2	1	1	2	2	1	1	1	2	2	1	1	1	2	2	2	2
44	2	1	1	2	2	1	1	1	2	2	1	1	1	2	2	2	2
43	2	2	2	1	1	1	1	1	2	2	1	1	1	2	2	2	2
42	1	2	2	1	1	1	1	1	1	2	2	2	1	1	1	1	1
41	1	2	2	1	2	2	2	1	1	2	2	2	1	1	1	2	2
40	2	2	1	1	2	2	2	1	1	2	2	2	1	2	2	2	2
39	2	2	1	2	2	2	2	2	2	1	1	1	1	2	2	2	2
38	1	1	1	2	2	2	2	2	2	1	1	1	1	2	2	1	1
37	2	2	1	2	2	2	2	2	2	1	1	1	1	2	2	1	1
36	1	1	1	2	2	2	2	2	2	1	1	2	2	2	2	1	1
35	1	1	1	2	2	2	2	2	2	1	1	2	2	2	2	1	1
34	2	2	1	2	2	2	2	2	2	1	1	2	2	1	1	1	1
33	2	2	1	2	1	1	1	2	2	1	1	2	2	1	1	1	1
32	2	2	1	2	1	1	1	1	1	1	1	2	2	1	2	2	1
31	2	2	1	2	2	2	1	1	2	2	2	2	1	1	2	2	1
30	2	2	1	1	2	2	1	1	2	2	2	2	1	1	2	2	1
29	2	2	1	1	2	2	1	2	2	2	2	2	2	1	2	2	2

28	2	2	1	1	1	1	1	2	2	2	2	2	2	1	2	2	2
27	2	2	1	1	1	1	2	2	2	2	2	2	1	1	2	2	2
26	2	2	1	1	1	1	2	2	2	2	2	2	1	1	2	2	2
25	2	1	1	1	1	1	2	2	2	2	2	2	1	2	2	2	2
24	2	2	2	2	2	1	1	2	2	1	1	1	1	2	2	2	2
23	2	2	2	2	2	1	1	2	2	1	1	1	1	2	2	2	2
22	1	2	2	2	2	1	1	2	2	1	1	1	1	2	2	2	1
21	2	1	2	2	2	2	1	2	2	2	2	2	1	2	2	2	1
20	2	1	1	1	2	2	2	2	2	2	2	2	1	1	1	1	1
19	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
18	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
17	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
16	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2
15	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2
14	2	2	1	1	2	2	2	2	2	2	2	2	1	1	1	1	1
13	2	2	1	1	2	2	2	2	2	2	2	2	1	1	1	1	1
12	2	2	1	1	2	2	2	2	2	2	2	2	1	1	1	1	1
11	1	1	1	1	1	1	1	2	2	2	2	2	1	1	1	1	1
10	1	1	1	1	1	1	1	2	2	2	2	2	1	1	1	1	1
9	2	2	2	1	1	1	1	2	2	2	2	2	1	1	1	1	1
8	1	1	2	2	2	1	1	2	2	2	2	2	1	1	1	1	1
7	2	2	2	2	2	1	1	1	1	2	2	2	1	1	1	1	1
6	2	2	2	2	2	1	2	1	1	2	2	2	1	1	1	1	1
5	1	2	2	2	1	1	2	1	1	2	2	2	2	2	2	2	2
4	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2
3	1	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2
2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
1	1	2	2	2	2	1	1	1	1	1	1	1	1	1	1	1	1

	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34
	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51
56	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
55	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
54	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
53	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
52	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
51	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
50	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
49	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
48	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
47	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
46	1	1	1	*	*	*	*	*	*	*	*	*	*	*	*	*	*
45	2	1	1	1	1	1	*	*	*	*	*	*	*	*	*	*	*
44	2	1	1	2	2	1	1	1	1	1	1	*	*	*	*	*	*
43	2	1	1	2	2	1	2	2	1	1	2	*	*	*	*	*	*
42	1	2	2	1	1	1	2	2	1	1	2	*	*	*	*	*	*
41	1	2	2	1	2	1	2	2	1	1	1	*	*	*	*	*	*
40	1	1	1	2	2	1	1	1	2	1	1	*	*	*	*	*	*
39	1	2	1	2	2	1	1	1	2	1	1	*	*	*	*	*	*
38	1	2	1	2	1	1	1	2	2	1	1	*	*	*	*	*	*
37	1	2	1	2	1	1	1	2	2	1	1	*	*	*	*	*	*
36	1	2	1	1	1	1	1	2	2	1	1	*	*	*	*	*	*

	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51
35	1	2	1	1	1	1	1	1	1	1	1	*	*	*	*	*	*
34	1	2	1	1	1	1	2	1	1	2	2	*	*	*	*	*	*
33	1	2	1	1	1	1	2	1	1	2	2	*	*	*	*	*	*
32	1	2	1	1	1	1	2	1	1	2	2	*	*	*	*	*	*
31	1	2	1	1	1	1	2	1	1	1	1	*	*	*	*	*	*
30	1	1	1	1	1	1	2	1	1	1	1	*	*	*	*	*	*
29	2	2	2	1	1	1	2	1	1	1	1	*	*	*	*	*	*
28	2	2	2	1	1	1	2	1	1	1	1	*	*	*	*	*	*
27	2	2	2	1	1	1	2	1	1	1	1	*	*	*	*	*	*
26	2	2	2	1	1	1	2	1	1	1	1	*	*	*	*	*	*
25	2	2	2	2	2	2	1	1	2	1	1	*	*	*	*	*	*
24	2	2	2	2	2	2	1	1	2	1	1	*	*	*	*	*	*
23	2	2	2	1	1	1	1	1	2	1	1	*	*	*	*	*	*
22	1	1	1	1	2	2	2	1	2	1	2	*	*	*	*	*	*
21	1	2	2	2	1	1	1	2	2	1	2	*	*	*	*	*	*
20	1	2	2	2	1	1	1	2	1	1	2	*	*	*	*	*	*
19	1	1	1	1	2	2	1	2	1	1	2	*	*	*	*	*	*
18	1	1	1	1	2	2	1	2	1	1	2	*	*	*	*	*	*
17	1	1	1	1	2	2	1	2	1	1	2	*	*	*	*	*	*
16	1	1	1	1	2	2	1	2	1	2	2	*	*	*	*	*	*
15	1	1	1	1	2	2	1	2	1	2	2	*	*	*	*	*	*
14	1	1	1	1	2	2	1	2	1	2	2	*	*	*	*	*	*
13	1	1	1	1	2	2	1	2	1	2	2	*	*	*	*	*	*
12	1	1	1	1	2	2	1	2	1	2	2	*	*	*	*	*	*
11	1	2	2	2	2	2	1	1	1	2	2	*	*	*	*	*	*
10	1	2	2	2	2	2	1	1	1	2	2	*	*	*	*	*	*
9	1	2	2	2	1	1	1	2	1	2	2	*	*	*	*	*	*
8	1	2	2	2	1	1	1	2	1	2	2	*	*	*	*	*	*
7	1	1	1	1	1	1	1	2	2	2	2	*	*	*	*	*	*
6	1	1	1	1	1	1	1	2	2	2	2	*	*	*	*	*	*
5	1	1	1	1	1	1	2	2	2	2	2	*	*	*	*	*	*
4	2	2	2	2	1	1	2	2	2	2	2	*	*	*	*	*	*
3	2	1	1	1	1	1	2	2	2	2	2	*	*	*	*	*	*
2	2	1	1	2	2	1	2	2	2	2	2	*	*	*	*	*	*
1	1	1	1	1	1	1	2	2	2	2	2	*	*	*	*	*	*

	52	53
56	*	*
55	*	*
54	*	*
53	*	*
52	*	*
51	*	*
50	*	*
49	*	*
48	*	*
47	*	*
46	*	*
45	*	*
44	*	*
43	*	*



***** PATHLINE & PARTICLE TRACKING DATA *****

Number of forward particles : 24

No. x-release y-release

1	3.48435E+02	3.97463E+03
2	3.48435E+02	3.97162E+03
3	3.46623E+02	3.97456E+03
4	3.48595E+02	3.97450E+03
5	3.48595E+02	3.97469E+03
6	3.48675E+02	3.97469E+03
7	3.48675E+02	3.97488E+03
8	3.48595E+02	3.97488E+03
9	3.48515E+02	3.97488E+03
10	3.48435E+02	3.97488E+03
11	3.48355E+02	3.97469E+03
12	3.48355E+02	3.97450E+03
13	3.48435E+02	3.97431E+03
14	3.48195E+02	3.97124E+03
15	3.48355E+02	3.97124E+03
16	3.48515E+02	3.97124E+03
17	3.48675E+02	3.97124E+03
18	3.48755E+02	3.97124E+03
19	3.48755E+02	3.97143E+03
20	3.48835E+02	3.97162E+03
21	3.48915E+02	3.97162E+03
22	3.48915E+02	3.97181E+03
23	3.48995E+02	3.97181E+03
24	3.48595E+02	3.97162E+03

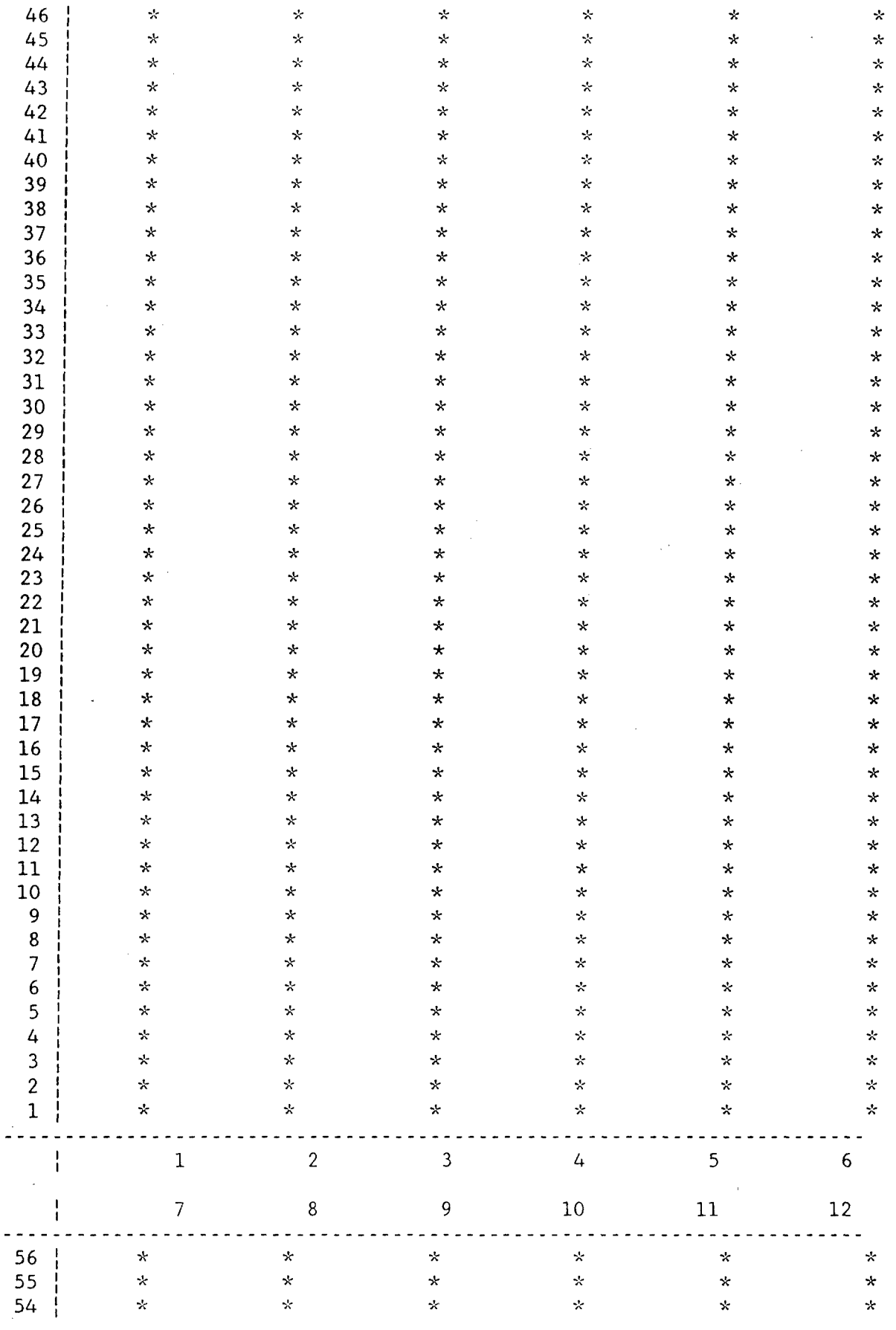
Number of reverse particles : 0

Particles released at wells :

Well-No.	Particles released
1	0
2	0

********** HYDRAULIC HEAD DISTRIBUTION **********

	1	2	3	4	5	6
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	*	*	*	*	*	*
49	*	*	*	*	*	*
48	*	*	*	*	*	*
47	*	*	*	*	*	*


46 45 44 43 42 41 40 39 38 37 36 35 34 33 32 31 30 29 28 27 26 25 24 23 22 21 20 19 18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1
1 2 3 4 5 6
7 8 9 10 11 12
56 55 54

53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	*	*	*	*	*	1.1354E-01
49	*	*	*	*	1.1354E-01	1.1354E-01
48	*	*	*	1.1430E-01	1.1392E-01	1.1374E-01
47	*	*	1.1430E-01	1.1430E-01	1.1416E-01	1.1401E-01
46	*	1.1472E-01	1.1468E-01	1.1457E-01	1.1445E-01	1.1432E-01
45	*	1.1510E-01	1.1506E-01	1.1494E-01	1.1481E-01	1.1468E-01
44	*	1.1582E-01	1.1556E-01	1.1536E-01	1.1518E-01	1.1502E-01
43	*	1.1582E-01	1.1566E-01	1.1551E-01	1.1537E-01	1.1524E-01
42	*	1.1582E-01	1.1571E-01	1.1561E-01	1.1550E-01	1.1540E-01
41	*	1.1582E-01	1.1575E-01	1.1568E-01	1.1561E-01	1.1554E-01
40	*	1.1582E-01	1.1579E-01	1.1575E-01	1.1571E-01	1.1568E-01
39	1.1582E-01	1.1582E-01	1.1583E-01	1.1583E-01	1.1582E-01	1.1581E-01
38	1.1602E-01	1.1600E-01	1.1599E-01	1.1598E-01	1.1597E-01	1.1597E-01
37	1.1605E-01	1.1606E-01	1.1608E-01	1.1609E-01	1.1611E-01	1.1612E-01
36	1.1598E-01	1.1608E-01	1.1614E-01	1.1619E-01	1.1622E-01	1.1625E-01
35	1.1613E-01	1.1620E-01	1.1625E-01	1.1630E-01	1.1634E-01	1.1637E-01
34	1.1628E-01	1.1633E-01	1.1638E-01	1.1642E-01	1.1645E-01	1.1648E-01
33	1.1643E-01	1.1647E-01	1.1651E-01	1.1655E-01	1.1658E-01	1.1660E-01
32	1.1661E-01	1.1663E-01	1.1665E-01	1.1668E-01	1.1671E-01	1.1673E-01
31	1.1674E-01	1.1677E-01	1.1679E-01	1.1682E-01	1.1684E-01	1.1686E-01
30	1.1689E-01	1.1691E-01	1.1693E-01	1.1695E-01	1.1697E-01	1.1700E-01
29	1.1704E-01	1.1705E-01	1.1707E-01	1.1709E-01	1.1711E-01	1.1713E-01
28	1.1720E-01	1.1719E-01	1.1720E-01	1.1722E-01	1.1724E-01	1.1726E-01
27	1.1727E-01	1.1729E-01	1.1732E-01	1.1734E-01	1.1737E-01	1.1739E-01
26	1.1735E-01	1.1742E-01	1.1746E-01	1.1750E-01	1.1753E-01	1.1756E-01
25	1.1765E-01	1.1768E-01	1.1772E-01	1.1774E-01	1.1777E-01	1.1780E-01
24	1.1796E-01	1.1798E-01	1.1800E-01	1.1802E-01	1.1804E-01	1.1806E-01
23	1.1826E-01	1.1828E-01	1.1830E-01	1.1831E-01	1.1833E-01	1.1835E-01
22	1.1857E-01	1.1859E-01	1.1861E-01	1.1863E-01	1.1865E-01	1.1867E-01
21	1.1887E-01	1.1891E-01	1.1893E-01	1.1895E-01	1.1897E-01	1.1899E-01
20	1.1926E-01	1.1926E-01	1.1927E-01	1.1928E-01	1.1930E-01	1.1931E-01
19	1.1953E-01	1.1953E-01	1.1954E-01	1.1955E-01	1.1955E-01	1.1956E-01
18	1.1972E-01	1.1972E-01	1.1973E-01	1.1974E-01	1.1974E-01	1.1975E-01
17	1.1989E-01	1.1989E-01	1.1990E-01	1.1991E-01	1.1991E-01	1.1992E-01
16	1.2006E-01	1.2006E-01	1.2007E-01	1.2007E-01	1.2008E-01	1.2009E-01
15	1.2023E-01	1.2023E-01	1.2023E-01	1.2024E-01	1.2025E-01	1.2025E-01
14	1.2039E-01	1.2039E-01	1.2039E-01	1.2040E-01	1.2041E-01	1.2042E-01
13	1.2055E-01	1.2055E-01	1.2055E-01	1.2056E-01	1.2057E-01	1.2057E-01
12	1.2070E-01	1.2070E-01	1.2071E-01	1.2071E-01	1.2072E-01	1.2073E-01
11	1.2085E-01	1.2085E-01	1.2086E-01	1.2087E-01	1.2087E-01	1.2088E-01
10	1.2100E-01	1.2100E-01	1.2101E-01	1.2101E-01	1.2102E-01	1.2103E-01
9	1.2118E-01	1.2118E-01	1.2119E-01	1.2119E-01	1.2121E-01	1.2122E-01
8	1.2141E-01	1.2141E-01	1.2142E-01	1.2143E-01	1.2144E-01	1.2146E-01
7	1.2164E-01	1.2164E-01	1.2165E-01	1.2167E-01	1.2169E-01	1.2172E-01
6	1.2181E-01	1.2182E-01	1.2183E-01	1.2185E-01	1.2188E-01	1.2192E-01
5	1.2193E-01	1.2194E-01	1.2196E-01	1.2198E-01	1.2202E-01	1.2206E-01
4	1.2200E-01	1.2201E-01	1.2203E-01	1.2206E-01	1.2210E-01	1.2215E-01
3	1.2199E-01	1.2200E-01	1.2202E-01	1.2204E-01	1.2208E-01	1.2213E-01
2	1.2192E-01	1.2192E-01	1.2192E-01	1.2192E-01	1.2192E-01	1.2192E-01
1	1.2200E-01	1.2201E-01	1.2202E-01	1.2205E-01	1.2210E-01	1.2216E-01

	7	8	9	10	11	12
	13	14	15	16	17	18
56	*	*	1.0973E-01	*	*	*
55	*	*	1.0973E-01	1.0973E-01	*	*
54	*	*	1.1156E-01	1.1064E-01	1.0973E-01	*
53	*	*	1.1217E-01	1.1097E-01	1.0973E-01	1.0973E-01
52	*	1.1247E-01	1.1247E-01	1.1153E-01	1.1061E-01	1.0973E-01
51	1.1278E-01	1.1278E-01	1.1225E-01	1.1168E-01	1.1107E-01	1.1042E-01
50	1.1316E-01	1.1282E-01	1.1246E-01	1.1209E-01	1.1172E-01	1.1137E-01
49	1.1330E-01	1.1304E-01	1.1278E-01	1.1252E-01	1.1226E-01	1.1202E-01
48	1.1355E-01	1.1335E-01	1.1314E-01	1.1295E-01	1.1275E-01	1.1255E-01
47	1.1385E-01	1.1368E-01	1.1352E-01	1.1335E-01	1.1319E-01	1.1303E-01
46	1.1419E-01	1.1404E-01	1.1390E-01	1.1376E-01	1.1362E-01	1.1348E-01
45	1.1455E-01	1.1441E-01	1.1428E-01	1.1416E-01	1.1403E-01	1.1391E-01
44	1.1488E-01	1.1474E-01	1.1461E-01	1.1450E-01	1.1439E-01	1.1427E-01
43	1.1511E-01	1.1499E-01	1.1487E-01	1.1477E-01	1.1468E-01	1.1458E-01
42	1.1530E-01	1.1519E-01	1.1509E-01	1.1502E-01	1.1495E-01	1.1487E-01
41	1.1548E-01	1.1541E-01	1.1534E-01	1.1528E-01	1.1522E-01	1.1517E-01
40	1.1564E-01	1.1560E-01	1.1556E-01	1.1552E-01	1.1548E-01	1.1544E-01
39	1.1579E-01	1.1578E-01	1.1576E-01	1.1574E-01	1.1572E-01	1.1570E-01
38	1.1597E-01	1.1596E-01	1.1596E-01	1.1595E-01	1.1595E-01	1.1595E-01
37	1.1613E-01	1.1614E-01	1.1615E-01	1.1616E-01	1.1617E-01	1.1617E-01
36	1.1627E-01	1.1629E-01	1.1631E-01	1.1633E-01	1.1634E-01	1.1636E-01
35	1.1639E-01	1.1642E-01	1.1644E-01	1.1646E-01	1.1648E-01	1.1649E-01
34	1.1651E-01	1.1653E-01	1.1656E-01	1.1658E-01	1.1660E-01	1.1662E-01
33	1.1663E-01	1.1666E-01	1.1668E-01	1.1670E-01	1.1672E-01	1.1674E-01
32	1.1676E-01	1.1678E-01	1.1681E-01	1.1683E-01	1.1685E-01	1.1687E-01
31	1.1689E-01	1.1691E-01	1.1694E-01	1.1696E-01	1.1698E-01	1.1700E-01
30	1.1702E-01	1.1704E-01	1.1707E-01	1.1709E-01	1.1711E-01	1.1713E-01
29	1.1715E-01	1.1717E-01	1.1720E-01	1.1722E-01	1.1724E-01	1.1727E-01
28	1.1728E-01	1.1731E-01	1.1733E-01	1.1736E-01	1.1738E-01	1.1740E-01
27	1.1742E-01	1.1744E-01	1.1747E-01	1.1749E-01	1.1751E-01	1.1753E-01
26	1.1759E-01	1.1762E-01	1.1764E-01	1.1766E-01	1.1768E-01	1.1771E-01
25	1.1782E-01	1.1785E-01	1.1787E-01	1.1789E-01	1.1791E-01	1.1793E-01
24	1.1808E-01	1.1811E-01	1.1813E-01	1.1815E-01	1.1817E-01	1.1819E-01
23	1.1837E-01	1.1839E-01	1.1841E-01	1.1843E-01	1.1845E-01	1.1847E-01
22	1.1869E-01	1.1871E-01	1.1873E-01	1.1874E-01	1.1876E-01	1.1878E-01
21	1.1901E-01	1.1903E-01	1.1904E-01	1.1906E-01	1.1908E-01	1.1909E-01
20	1.1932E-01	1.1934E-01	1.1935E-01	1.1936E-01	1.1938E-01	1.1939E-01
19	1.1957E-01	1.1958E-01	1.1960E-01	1.1961E-01	1.1962E-01	1.1964E-01
18	1.1976E-01	1.1977E-01	1.1979E-01	1.1980E-01	1.1981E-01	1.1982E-01
17	1.1993E-01	1.1994E-01	1.1995E-01	1.1996E-01	1.1998E-01	1.1999E-01
16	1.2010E-01	1.2011E-01	1.2012E-01	1.2013E-01	1.2014E-01	1.2015E-01
15	1.2026E-01	1.2027E-01	1.2029E-01	1.2030E-01	1.2031E-01	1.2032E-01
14	1.2043E-01	1.2044E-01	1.2045E-01	1.2046E-01	1.2048E-01	1.2049E-01
13	1.2059E-01	1.2060E-01	1.2061E-01	1.2063E-01	1.2064E-01	1.2066E-01
12	1.2074E-01	1.2076E-01	1.2077E-01	1.2079E-01	1.2081E-01	1.2083E-01
11	1.2090E-01	1.2091E-01	1.2093E-01	1.2095E-01	1.2097E-01	1.2099E-01
10	1.2105E-01	1.2107E-01	1.2109E-01	1.2111E-01	1.2114E-01	1.2116E-01
9	1.2124E-01	1.2126E-01	1.2129E-01	1.2131E-01	1.2134E-01	1.2137E-01
8	1.2149E-01	1.2152E-01	1.2155E-01	1.2158E-01	1.2161E-01	1.2165E-01
7	1.2175E-01	1.2179E-01	1.2183E-01	1.2187E-01	1.2191E-01	1.2197E-01

6	1.2196E-01	1.2201E-01	1.2206E-01	1.2212E-01	1.2218E-01	1.2225E-01
5	1.2211E-01	1.2218E-01	1.2225E-01	1.2233E-01	1.2242E-01	1.2251E-01
4	1.2222E-01	1.2230E-01	1.2241E-01	1.2252E-01	1.2264E-01	1.2277E-01
3	1.2220E-01	1.2231E-01	1.2247E-01	1.2266E-01	1.2285E-01	1.2304E-01
2	1.2192E-01	1.2192E-01	1.2230E-01	1.2268E-01	1.2306E-01	1.2344E-01
1	1.2225E-01	1.2239E-01	1.2259E-01	1.2282E-01	1.2306E-01	1.2330E-01

	13	14	15	16	17	18
	19	20	21	22	23	24
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	1.0973E-01	1.0973E-01	1.0973E-01	*	*	*
51	1.0973E-01	1.0973E-01	1.0973E-01	1.0973E-01	1.0981E-01	*
50	1.1106E-01	1.1083E-01	1.1065E-01	1.1051E-01	1.1040E-01	1.1073E-01
49	1.1179E-01	1.1157E-01	1.1135E-01	1.1115E-01	1.1095E-01	1.1073E-01
48	1.1237E-01	1.1218E-01	1.1199E-01	1.1180E-01	1.1162E-01	1.1144E-01
47	1.1287E-01	1.1271E-01	1.1254E-01	1.1238E-01	1.1222E-01	1.1207E-01
46	1.1334E-01	1.1319E-01	1.1306E-01	1.1291E-01	1.1277E-01	1.1263E-01
45	1.1378E-01	1.1366E-01	1.1354E-01	1.1341E-01	1.1329E-01	1.1317E-01
44	1.1416E-01	1.1405E-01	1.1395E-01	1.1385E-01	1.1374E-01	1.1364E-01
43	1.1449E-01	1.1439E-01	1.1431E-01	1.1422E-01	1.1413E-01	1.1404E-01
42	1.1480E-01	1.1473E-01	1.1466E-01	1.1459E-01	1.1452E-01	1.1445E-01
41	1.1511E-01	1.1506E-01	1.1501E-01	1.1496E-01	1.1491E-01	1.1486E-01
40	1.1541E-01	1.1537E-01	1.1534E-01	1.1531E-01	1.1528E-01	1.1525E-01
39	1.1568E-01	1.1566E-01	1.1565E-01	1.1563E-01	1.1562E-01	1.1560E-01
38	1.1594E-01	1.1594E-01	1.1594E-01	1.1594E-01	1.1594E-01	1.1594E-01
37	1.1618E-01	1.1619E-01	1.1620E-01	1.1621E-01	1.1622E-01	1.1624E-01
36	1.1637E-01	1.1639E-01	1.1640E-01	1.1642E-01	1.1644E-01	1.1646E-01
35	1.1651E-01	1.1653E-01	1.1655E-01	1.1657E-01	1.1659E-01	1.1662E-01
34	1.1664E-01	1.1666E-01	1.1668E-01	1.1670E-01	1.1673E-01	1.1675E-01
33	1.1676E-01	1.1679E-01	1.1681E-01	1.1683E-01	1.1686E-01	1.1689E-01
32	1.1690E-01	1.1692E-01	1.1694E-01	1.1696E-01	1.1699E-01	1.1702E-01
31	1.1703E-01	1.1705E-01	1.1707E-01	1.1709E-01	1.1712E-01	1.1715E-01
30	1.1716E-01	1.1718E-01	1.1720E-01	1.1723E-01	1.1725E-01	1.1728E-01
29	1.1729E-01	1.1731E-01	1.1733E-01	1.1736E-01	1.1738E-01	1.1741E-01
28	1.1742E-01	1.1744E-01	1.1747E-01	1.1749E-01	1.1751E-01	1.1754E-01
27	1.1756E-01	1.1758E-01	1.1760E-01	1.1762E-01	1.1764E-01	1.1767E-01
26	1.1773E-01	1.1775E-01	1.1777E-01	1.1779E-01	1.1781E-01	1.1783E-01
25	1.1795E-01	1.1797E-01	1.1799E-01	1.1801E-01	1.1803E-01	1.1805E-01
24	1.1821E-01	1.1822E-01	1.1824E-01	1.1826E-01	1.1828E-01	1.1829E-01
23	1.1849E-01	1.1850E-01	1.1852E-01	1.1853E-01	1.1855E-01	1.1856E-01
22	1.1879E-01	1.1881E-01	1.1882E-01	1.1884E-01	1.1885E-01	1.1887E-01
21	1.1911E-01	1.1912E-01	1.1913E-01	1.1915E-01	1.1916E-01	1.1917E-01
20	1.1941E-01	1.1942E-01	1.1943E-01	1.1945E-01	1.1946E-01	1.1947E-01
19	1.1965E-01	1.1966E-01	1.1968E-01	1.1969E-01	1.1970E-01	1.1971E-01
18	1.1984E-01	1.1985E-01	1.1986E-01	1.1988E-01	1.1989E-01	1.1990E-01
17	1.2000E-01	1.2002E-01	1.2003E-01	1.2005E-01	1.2006E-01	1.2007E-01
16	1.2017E-01	1.2018E-01	1.2020E-01	1.2022E-01	1.2023E-01	1.2024E-01
15	1.2034E-01	1.2036E-01	1.2037E-01	1.2039E-01	1.2040E-01	1.2042E-01
14	1.2051E-01	1.2053E-01	1.2054E-01	1.2056E-01	1.2058E-01	1.2059E-01

13	1.2068E-01	1.2070E-01	1.2072E-01	1.2074E-01	1.2075E-01	1.2077E-01
12	1.2085E-01	1.2087E-01	1.2089E-01	1.2091E-01	1.2093E-01	1.2095E-01
11	1.2102E-01	1.2104E-01	1.2106E-01	1.2108E-01	1.2111E-01	1.2114E-01
10	1.2119E-01	1.2121E-01	1.2124E-01	1.2126E-01	1.2129E-01	1.2132E-01
9	1.2140E-01	1.2143E-01	1.2146E-01	1.2149E-01	1.2153E-01	1.2157E-01
8	1.2169E-01	1.2173E-01	1.2177E-01	1.2182E-01	1.2186E-01	1.2191E-01
7	1.2202E-01	1.2208E-01	1.2213E-01	1.2219E-01	1.2225E-01	1.2231E-01
6	1.2232E-01	1.2240E-01	1.2247E-01	1.2254E-01	1.2262E-01	1.2270E-01
5	1.2260E-01	1.2270E-01	1.2279E-01	1.2289E-01	1.2298E-01	1.2308E-01
4	1.2290E-01	1.2302E-01	1.2314E-01	1.2327E-01	1.2340E-01	1.2352E-01
3	1.2322E-01	1.2339E-01	1.2355E-01	1.2371E-01	1.2387E-01	1.2403E-01
2	1.2363E-01	1.2383E-01	1.2402E-01	1.2421E-01	1.2440E-01	1.2459E-01
1	1.2352E-01	1.2373E-01	1.2393E-01	1.2413E-01	1.2433E-01	1.2453E-01

	19	20	21	22	23	24
	25	26	27	28	29	30
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	1.1075E-01	*	*	*	*	*
49	1.1090E-01	1.1107E-01	1.1107E-01	*	*	*
48	1.1126E-01	1.1107E-01	1.1115E-01	1.1124E-01	1.1132E-01	1.1140E-01
47	1.1191E-01	1.1177E-01	1.1168E-01	1.1160E-01	1.1151E-01	1.1140E-01
46	1.1249E-01	1.1237E-01	1.1228E-01	1.1220E-01	1.1212E-01	1.1204E-01
45	1.1305E-01	1.1295E-01	1.1287E-01	1.1281E-01	1.1275E-01	1.1269E-01
44	1.1354E-01	1.1345E-01	1.1339E-01	1.1333E-01	1.1328E-01	1.1323E-01
43	1.1396E-01	1.1389E-01	1.1384E-01	1.1380E-01	1.1376E-01	1.1372E-01
42	1.1438E-01	1.1433E-01	1.1429E-01	1.1426E-01	1.1424E-01	1.1421E-01
41	1.1481E-01	1.1478E-01	1.1475E-01	1.1473E-01	1.1472E-01	1.1470E-01
40	1.1522E-01	1.1520E-01	1.1518E-01	1.1517E-01	1.1516E-01	1.1515E-01
39	1.1559E-01	1.1559E-01	1.1558E-01	1.1558E-01	1.1558E-01	1.1557E-01
38	1.1594E-01	1.1595E-01	1.1596E-01	1.1596E-01	1.1597E-01	1.1598E-01
37	1.1625E-01	1.1627E-01	1.1629E-01	1.1630E-01	1.1631E-01	1.1633E-01
36	1.1648E-01	1.1651E-01	1.1653E-01	1.1654E-01	1.1656E-01	1.1658E-01
35	1.1664E-01	1.1667E-01	1.1669E-01	1.1671E-01	1.1673E-01	1.1675E-01
34	1.1678E-01	1.1681E-01	1.1683E-01	1.1685E-01	1.1687E-01	1.1690E-01
33	1.1692E-01	1.1694E-01	1.1697E-01	1.1699E-01	1.1701E-01	1.1703E-01
32	1.1705E-01	1.1707E-01	1.1709E-01	1.1711E-01	1.1713E-01	1.1715E-01
31	1.1717E-01	1.1720E-01	1.1722E-01	1.1724E-01	1.1725E-01	1.1727E-01
30	1.1730E-01	1.1733E-01	1.1734E-01	1.1736E-01	1.1737E-01	1.1739E-01
29	1.1743E-01	1.1745E-01	1.1747E-01	1.1748E-01	1.1750E-01	1.1751E-01
28	1.1756E-01	1.1758E-01	1.1759E-01	1.1761E-01	1.1762E-01	1.1763E-01
27	1.1769E-01	1.1771E-01	1.1772E-01	1.1773E-01	1.1774E-01	1.1775E-01
26	1.1785E-01	1.1787E-01	1.1788E-01	1.1789E-01	1.1790E-01	1.1791E-01
25	1.1806E-01	1.1808E-01	1.1809E-01	1.1810E-01	1.1811E-01	1.1812E-01
24	1.1831E-01	1.1832E-01	1.1833E-01	1.1834E-01	1.1835E-01	1.1836E-01
23	1.1858E-01	1.1859E-01	1.1860E-01	1.1861E-01	1.1861E-01	1.1862E-01
22	1.1888E-01	1.1889E-01	1.1890E-01	1.1890E-01	1.1891E-01	1.1891E-01
21	1.1919E-01	1.1920E-01	1.1921E-01	1.1921E-01	1.1922E-01	1.1922E-01

20	1.1948E-01	1.1949E-01	1.1950E-01	1.1951E-01	1.1951E-01	1.1952E-01
19	1.1973E-01	1.1974E-01	1.1975E-01	1.1975E-01	1.1976E-01	1.1976E-01
18	1.1992E-01	1.1993E-01	1.1993E-01	1.1994E-01	1.1994E-01	1.1995E-01
17	1.2008E-01	1.2009E-01	1.2010E-01	1.2011E-01	1.2011E-01	1.2012E-01
16	1.2026E-01	1.2027E-01	1.2028E-01	1.2028E-01	1.2029E-01	1.2029E-01
15	1.2043E-01	1.2045E-01	1.2045E-01	1.2046E-01	1.2047E-01	1.2048E-01
14	1.2061E-01	1.2063E-01	1.2064E-01	1.2065E-01	1.2065E-01	1.2066E-01
13	1.2079E-01	1.2081E-01	1.2082E-01	1.2083E-01	1.2084E-01	1.2085E-01
12	1.2098E-01	1.2100E-01	1.2101E-01	1.2102E-01	1.2103E-01	1.2104E-01
11	1.2116E-01	1.2119E-01	1.2120E-01	1.2121E-01	1.2123E-01	1.2124E-01
10	1.2135E-01	1.2138E-01	1.2140E-01	1.2141E-01	1.2143E-01	1.2144E-01
9	1.2160E-01	1.2163E-01	1.2165E-01	1.2167E-01	1.2168E-01	1.2170E-01
8	1.2195E-01	1.2199E-01	1.2201E-01	1.2203E-01	1.2206E-01	1.2208E-01
7	1.2237E-01	1.2242E-01	1.2245E-01	1.2248E-01	1.2251E-01	1.2253E-01
6	1.2277E-01	1.2284E-01	1.2289E-01	1.2292E-01	1.2296E-01	1.2300E-01
5	1.2318E-01	1.2327E-01	1.2333E-01	1.2338E-01	1.2343E-01	1.2348E-01
4	1.2365E-01	1.2377E-01	1.2385E-01	1.2392E-01	1.2398E-01	1.2404E-01
3	1.2420E-01	1.2435E-01	1.2446E-01	1.2454E-01	1.2462E-01	1.2470E-01
2	1.2478E-01	1.2497E-01	1.2509E-01	1.2520E-01	1.2532E-01	1.2544E-01
1	1.2473E-01	1.2492E-01	1.2505E-01	1.2516E-01	1.2526E-01	1.2536E-01

	25	26	27	28	29	30
	31	32	33	34	35	36
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	*	*	*	*	*	*
49	*	*	*	*	*	*
48	*	*	*	*	*	*
47	1.1153E-01	1.1165E-01	1.1177E-01	*	*	*
46	1.1196E-01	1.1187E-01	1.1177E-01	1.1185E-01	1.1194E-01	1.1202E-01
45	1.1263E-01	1.1257E-01	1.1252E-01	1.1247E-01	1.1242E-01	1.1237E-01
44	1.1318E-01	1.1313E-01	1.1308E-01	1.1303E-01	1.1298E-01	1.1292E-01
43	1.1368E-01	1.1364E-01	1.1361E-01	1.1357E-01	1.1354E-01	1.1351E-01
42	1.1418E-01	1.1415E-01	1.1412E-01	1.1410E-01	1.1407E-01	1.1405E-01
41	1.1468E-01	1.1467E-01	1.1466E-01	1.1464E-01	1.1464E-01	1.1463E-01
40	1.1515E-01	1.1514E-01	1.1513E-01	1.1513E-01	1.1512E-01	1.1512E-01
39	1.1557E-01	1.1557E-01	1.1557E-01	1.1557E-01	1.1557E-01	1.1557E-01
38	1.1598E-01	1.1599E-01	1.1600E-01	1.1601E-01	1.1602E-01	1.1603E-01
37	1.1635E-01	1.1637E-01	1.1639E-01	1.1641E-01	1.1643E-01	1.1646E-01
36	1.1660E-01	1.1663E-01	1.1666E-01	1.1669E-01	1.1672E-01	1.1676E-01
35	1.1678E-01	1.1680E-01	1.1683E-01	1.1686E-01	1.1690E-01	1.1693E-01
34	1.1692E-01	1.1695E-01	1.1697E-01	1.1700E-01	1.1703E-01	1.1706E-01
33	1.1705E-01	1.1707E-01	1.1710E-01	1.1712E-01	1.1715E-01	1.1717E-01
32	1.1717E-01	1.1719E-01	1.1721E-01	1.1723E-01	1.1725E-01	1.1727E-01
31	1.1729E-01	1.1731E-01	1.1732E-01	1.1734E-01	1.1736E-01	1.1738E-01
30	1.1740E-01	1.1742E-01	1.1744E-01	1.1745E-01	1.1746E-01	1.1748E-01
29	1.1752E-01	1.1754E-01	1.1755E-01	1.1756E-01	1.1757E-01	1.1759E-01
28	1.1764E-01	1.1765E-01	1.1766E-01	1.1767E-01	1.1769E-01	1.1770E-01

27	1.1776E-01	1.1777E-01	1.1778E-01	1.1779E-01	1.1780E-01	1.1781E-01
26	1.1792E-01	1.1793E-01	1.1794E-01	1.1794E-01	1.1795E-01	1.1796E-01
25	1.1813E-01	1.1813E-01	1.1814E-01	1.1815E-01	1.1816E-01	1.1816E-01
24	1.1836E-01	1.1837E-01	1.1838E-01	1.1838E-01	1.1839E-01	1.1839E-01
23	1.1863E-01	1.1863E-01	1.1864E-01	1.1864E-01	1.1865E-01	1.1865E-01
22	1.1892E-01	1.1892E-01	1.1893E-01	1.1893E-01	1.1894E-01	1.1895E-01
21	1.1923E-01	1.1923E-01	1.1924E-01	1.1924E-01	1.1925E-01	1.1925E-01
20	1.1952E-01	1.1953E-01	1.1953E-01	1.1954E-01	1.1954E-01	1.1955E-01
19	1.1977E-01	1.1977E-01	1.1978E-01	1.1978E-01	1.1978E-01	1.1979E-01
18	1.1995E-01	1.1996E-01	1.1996E-01	1.1997E-01	1.1997E-01	1.1998E-01
17	1.2012E-01	1.2013E-01	1.2014E-01	1.2014E-01	1.2015E-01	1.2015E-01
16	1.2030E-01	1.2031E-01	1.2031E-01	1.2032E-01	1.2032E-01	1.2033E-01
15	1.2048E-01	1.2049E-01	1.2050E-01	1.2050E-01	1.2051E-01	1.2051E-01
14	1.2067E-01	1.2068E-01	1.2068E-01	1.2069E-01	1.2070E-01	1.2070E-01
13	1.2086E-01	1.2087E-01	1.2087E-01	1.2088E-01	1.2089E-01	1.2090E-01
12	1.2105E-01	1.2106E-01	1.2107E-01	1.2108E-01	1.2108E-01	1.2109E-01
11	1.2125E-01	1.2126E-01	1.2127E-01	1.2128E-01	1.2128E-01	1.2129E-01
10	1.2145E-01	1.2146E-01	1.2147E-01	1.2148E-01	1.2149E-01	1.2150E-01
9	1.2171E-01	1.2173E-01	1.2174E-01	1.2175E-01	1.2177E-01	1.2178E-01
8	1.2210E-01	1.2212E-01	1.2213E-01	1.2215E-01	1.2217E-01	1.2218E-01
7	1.2256E-01	1.2259E-01	1.2261E-01	1.2263E-01	1.2265E-01	1.2268E-01
6	1.2304E-01	1.2307E-01	1.2310E-01	1.2313E-01	1.2316E-01	1.2319E-01
5	1.2352E-01	1.2357E-01	1.2361E-01	1.2365E-01	1.2368E-01	1.2372E-01
4	1.2410E-01	1.2415E-01	1.2421E-01	1.2426E-01	1.2431E-01	1.2436E-01
3	1.2478E-01	1.2486E-01	1.2494E-01	1.2502E-01	1.2510E-01	1.2517E-01
2	1.2555E-01	1.2567E-01	1.2579E-01	1.2591E-01	1.2602E-01	1.2614E-01
1	1.2545E-01	1.2556E-01	1.2566E-01	1.2576E-01	1.2585E-01	1.2594E-01

	31	32	33	34	35	36
	37	38	39	40	41	42
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	*	*	*	*	*	*
49	*	*	*	*	*	*
48	*	*	*	*	*	*
47	*	*	*	*	*	*
46	1.1215E-01	*	*	*	*	*
45	1.1230E-01	1.1222E-01	1.1233E-01	1.1244E-01	*	*
44	1.1286E-01	1.1279E-01	1.1271E-01	1.1261E-01	1.1261E-01	1.1278E-01
43	1.1348E-01	1.1345E-01	1.1342E-01	1.1340E-01	1.1340E-01	1.1345E-01
42	1.1402E-01	1.1400E-01	1.1397E-01	1.1393E-01	1.1386E-01	1.1375E-01
41	1.1463E-01	1.1463E-01	1.1463E-01	1.1464E-01	1.1468E-01	1.1476E-01
40	1.1511E-01	1.1511E-01	1.1511E-01	1.1511E-01	1.1511E-01	1.1511E-01
39	1.1557E-01	1.1557E-01	1.1557E-01	1.1556E-01	1.1556E-01	1.1554E-01
38	1.1604E-01	1.1605E-01	1.1606E-01	1.1607E-01	1.1608E-01	1.1607E-01
37	1.1649E-01	1.1652E-01	1.1656E-01	1.1661E-01	1.1669E-01	1.1679E-01
36	1.1679E-01	1.1684E-01	1.1689E-01	1.1696E-01	1.1709E-01	1.1729E-01
35	1.1697E-01	1.1701E-01	1.1706E-01	1.1713E-01	1.1724E-01	1.1740E-01

34	1.1710E-01	1.1713E-01	1.1717E-01	1.1723E-01	1.1732E-01	1.1742E-01
33	1.1720E-01	1.1723E-01	1.1726E-01	1.1731E-01	1.1737E-01	1.1745E-01
32	1.1730E-01	1.1733E-01	1.1735E-01	1.1739E-01	1.1743E-01	1.1748E-01
31	1.1740E-01	1.1742E-01	1.1744E-01	1.1746E-01	1.1750E-01	1.1753E-01
30	1.1750E-01	1.1751E-01	1.1753E-01	1.1755E-01	1.1757E-01	1.1760E-01
29	1.1760E-01	1.1761E-01	1.1762E-01	1.1764E-01	1.1765E-01	1.1768E-01
28	1.1771E-01	1.1772E-01	1.1773E-01	1.1774E-01	1.1775E-01	1.1777E-01
27	1.1782E-01	1.1783E-01	1.1784E-01	1.1785E-01	1.1786E-01	1.1787E-01
26	1.1797E-01	1.1797E-01	1.1798E-01	1.1799E-01	1.1800E-01	1.1801E-01
25	1.1817E-01	1.1818E-01	1.1818E-01	1.1819E-01	1.1819E-01	1.1820E-01
24	1.1840E-01	1.1841E-01	1.1841E-01	1.1842E-01	1.1842E-01	1.1843E-01
23	1.1866E-01	1.1866E-01	1.1867E-01	1.1867E-01	1.1868E-01	1.1869E-01
22	1.1895E-01	1.1895E-01	1.1896E-01	1.1896E-01	1.1897E-01	1.1898E-01
21	1.1925E-01	1.1926E-01	1.1926E-01	1.1927E-01	1.1927E-01	1.1928E-01
20	1.1955E-01	1.1955E-01	1.1956E-01	1.1956E-01	1.1956E-01	1.1957E-01
19	1.1979E-01	1.1979E-01	1.1980E-01	1.1980E-01	1.1980E-01	1.1980E-01
18	1.1998E-01	1.1998E-01	1.1998E-01	1.1998E-01	1.1999E-01	1.1999E-01
17	1.2015E-01	1.2016E-01	1.2016E-01	1.2016E-01	1.2016E-01	1.2016E-01
16	1.2033E-01	1.2034E-01	1.2034E-01	1.2034E-01	1.2035E-01	1.2035E-01
15	1.2052E-01	1.2052E-01	1.2053E-01	1.2053E-01	1.2053E-01	1.2054E-01
14	1.2071E-01	1.2071E-01	1.2072E-01	1.2072E-01	1.2073E-01	1.2073E-01
13	1.2090E-01	1.2091E-01	1.2091E-01	1.2092E-01	1.2093E-01	1.2093E-01
12	1.2110E-01	1.2111E-01	1.2111E-01	1.2112E-01	1.2113E-01	1.2114E-01
11	1.2130E-01	1.2131E-01	1.2132E-01	1.2133E-01	1.2134E-01	1.2135E-01
10	1.2151E-01	1.2152E-01	1.2153E-01	1.2154E-01	1.2156E-01	1.2157E-01
9	1.2179E-01	1.2180E-01	1.2182E-01	1.2183E-01	1.2184E-01	1.2186E-01
8	1.2220E-01	1.2221E-01	1.2223E-01	1.2224E-01	1.2226E-01	1.2228E-01
7	1.2270E-01	1.2272E-01	1.2274E-01	1.2276E-01	1.2278E-01	1.2280E-01
6	1.2321E-01	1.2324E-01	1.2326E-01	1.2329E-01	1.2331E-01	1.2333E-01
5	1.2375E-01	1.2379E-01	1.2382E-01	1.2385E-01	1.2389E-01	1.2392E-01
4	1.2441E-01	1.2446E-01	1.2450E-01	1.2455E-01	1.2461E-01	1.2467E-01
3	1.2524E-01	1.2531E-01	1.2537E-01	1.2543E-01	1.2551E-01	1.2560E-01
2	1.2626E-01	1.2637E-01	1.2649E-01	1.2649E-01	1.2649E-01	1.2664E-01
1	1.2603E-01	1.2611E-01	1.2618E-01	1.2626E-01	1.2635E-01	1.2646E-01

	37	38	39	40	41	42
	43	44	45	46	47	48
56	*	*	*	*	*	*
55	*	*	*	*	*	*
54	*	*	*	*	*	*
53	*	*	*	*	*	*
52	*	*	*	*	*	*
51	*	*	*	*	*	*
50	*	*	*	*	*	*
49	*	*	*	*	*	*
48	*	*	*	*	*	*
47	*	*	*	*	*	*
46	*	*	*	*	*	*
45	*	*	*	*	*	*
44	1.1360E-01	1.1442E-01	1.1491E-01	*	*	*
43	1.1357E-01	1.1376E-01	1.1389E-01	*	*	*
42	1.1354E-01	1.1354E-01	1.1372E-01	*	*	*

41	1.1492E-01	1.1521E-01	1.1506E-01	*	*	*
40	1.1510E-01	1.1506E-01	1.1506E-01	*	*	*
39	1.1552E-01	1.1549E-01	1.1544E-01	*	*	*
38	1.1605E-01	1.1597E-01	1.1582E-01	*	*	*
37	1.1690E-01	1.1702E-01	1.1712E-01	*	*	*
36	1.1757E-01	1.1803E-01	1.1887E-01	*	*	*
35	1.1758E-01	1.1780E-01	1.1802E-01	*	*	*
34	1.1753E-01	1.1764E-01	1.1773E-01	*	*	*
33	1.1751E-01	1.1757E-01	1.1761E-01	*	*	*
32	1.1753E-01	1.1756E-01	1.1758E-01	*	*	*
31	1.1756E-01	1.1759E-01	1.1760E-01	*	*	*
30	1.1762E-01	1.1764E-01	1.1765E-01	*	*	*
29	1.1769E-01	1.1771E-01	1.1771E-01	*	*	*
28	1.1778E-01	1.1779E-01	1.1780E-01	*	*	*
27	1.1788E-01	1.1789E-01	1.1789E-01	*	*	*
26	1.1801E-01	1.1802E-01	1.1803E-01	*	*	*
25	1.1821E-01	1.1821E-01	1.1822E-01	*	*	*
24	1.1844E-01	1.1844E-01	1.1844E-01	*	*	*
23	1.1869E-01	1.1870E-01	1.1870E-01	*	*	*
22	1.1898E-01	1.1898E-01	1.1898E-01	*	*	*
21	1.1928E-01	1.1928E-01	1.1928E-01	*	*	*
20	1.1957E-01	1.1957E-01	1.1957E-01	*	*	*
19	1.1981E-01	1.1981E-01	1.1981E-01	*	*	*
18	1.1999E-01	1.1999E-01	1.1999E-01	*	*	*
17	1.2016E-01	1.2015E-01	1.2014E-01	*	*	*
16	1.2034E-01	1.2033E-01	1.2032E-01	*	*	*
15	1.2054E-01	1.2054E-01	1.2053E-01	*	*	*
14	1.2074E-01	1.2074E-01	1.2074E-01	*	*	*
13	1.2094E-01	1.2094E-01	1.2095E-01	*	*	*
12	1.2115E-01	1.2115E-01	1.2116E-01	*	*	*
11	1.2136E-01	1.2137E-01	1.2137E-01	*	*	*
10	1.2158E-01	1.2159E-01	1.2159E-01	*	*	*
9	1.2187E-01	1.2188E-01	1.2188E-01	*	*	*
8	1.2229E-01	1.2230E-01	1.2231E-01	*	*	*
7	1.2282E-01	1.2283E-01	1.2284E-01	*	*	*
6	1.2336E-01	1.2338E-01	1.2340E-01	*	*	*
5	1.2396E-01	1.2399E-01	1.2401E-01	*	*	*
4	1.2473E-01	1.2477E-01	1.2479E-01	*	*	*
3	1.2568E-01	1.2575E-01	1.2579E-01	*	*	*
2	1.2680E-01	1.2695E-01	1.2710E-01	*	*	*
1	1.2656E-01	1.2664E-01	1.2669E-01	*	*	*

	43	44	45	46	47	48

	49	50	51	52	53
56	*	*	*	*	*
55	*	*	*	*	*
54	*	*	*	*	*
53	*	*	*	*	*
52	*	*	*	*	*
51	*	*	*	*	*
50	*	*	*	*	*
49	*	*	*	*	*

48	*	*	*	*	*
47	*	*	*	*	*
46	*	*	*	*	*
45	*	*	*	*	*
44	*	*	*	*	*
43	*	*	*	*	*
42	*	*	*	*	*
41	*	*	*	*	*
40	*	*	*	*	*
39	*	*	*	*	*
38	*	*	*	*	*
37	*	*	*	*	*
36	*	*	*	*	*
35	*	*	*	*	*
34	*	*	*	*	*
33	*	*	*	*	*
32	*	*	*	*	*
31	*	*	*	*	*
30	*	*	*	*	*
29	*	*	*	*	*
28	*	*	*	*	*
27	*	*	*	*	*
26	*	*	*	*	*
25	*	*	*	*	*
24	*	*	*	*	*
23	*	*	*	*	*
22	*	*	*	*	*
21	*	*	*	*	*
20	*	*	*	*	*
19	*	*	*	*	*
18	*	*	*	*	*
17	*	*	*	*	*
16	*	*	*	*	*
15	*	*	*	*	*
14	*	*	*	*	*
13	*	*	*	*	*
12	*	*	*	*	*
11	*	*	*	*	*
10	*	*	*	*	*
9	*	*	*	*	*
8	*	*	*	*	*
7	*	*	*	*	*
6	*	*	*	*	*
5	*	*	*	*	*
4	*	*	*	*	*
3	*	*	*	*	*
2	*	*	*	*	*
1	*	*	*	*	*
	49	50	51	52	53

********** End of logbook **********

APPENDIX O

AIR EMISSIONS AND DISPERSION MODELING: TECHNICAL DISCUSSION

APPENDIX O

AIR EMISSIONS AND DISPERSION MODELING:
TECHNICAL DISCUSSION

O.1 METHODOLOGY

The following appendix describes the air dispersion models used to predict air concentrations of dust emissions from the Open Burning Ground (OBG) of the Milan Army Ammunition Plant (MAAP). Contaminants were detected in the surface soils of the OBG. Because soil surfaces at the site are not completely covered by vegetation, wind entrainment of dust particles is possible. As a result, inhalation of respirable particulate matter was considered to be a possible exposure pathway for on-site workers in the OBG and for nearby residents. Estimations of emissions due to wind erosion are based on Cowherd et. al. (1984). These emissions were input into the appropriate air dispersion models to estimate on- and off-site concentrations. The Box model was used to estimate air concentrations for individuals working in the OBG, and the Fugitive Dust Model (FDM) was used to estimate air concentrations for residents along the eastern border of MAAP, approximately one mile from the OBG. These models are described in detail in the following sections.

O.2 WIND EROSION EMISSIONS MODEL

Airborne particulate matter with an aerodynamic diameter less than or equal to 10 μm, referred to as PM_{10}, is respirable and, when contaminated, can contribute to inhalation exposure (Cowherd et al. 1984). The methodologies described by Cowherd et al. (1984) were used to calculate the emission rates of PM_{10}. Emission rates were determined from emission factors, the source extent, and the mass fraction of the chemicals in the soil. The PM_{10} emission factors employed in this method were empirically derived through regression analysis for field test data (Cowherd et al. 1984). The calculated PM_{10} emission rates were then linked to the appropriate atmospheric dispersion models which predicted the ambient air concentrations of respirable particulate matter on- and off-site.

The first step in estimating PM_{10} emissions associated with wind erosion of unvegetated portions of the contaminated site is the classification of the soil surface material. The soil surface is classified as having either a "limited reservoir" or an "unlimited reservoir" of erodible surface particles. Different equations are used to determine the wind erosion from these two classes of soil surfaces. Sandy soils with sparse vegetation, like the soil surface at the OBG, represent an "unlimited reservoir" of erodible particles. Thus the OBG was assumed to be an "unlimited reservoir."

Entrainment of particle matter is dependent on wind speed. For a given soil type there is a threshold wind velocity which must be attained to initiate entrainment. For the OBG area, this threshold wind velocity was determined following the method described by Cowherd et al. (1984). By specifying the soil aggregate particle size distribution mode, the threshold surface friction velocity can be determined from Figure 3-4 presented in Cowherd et al. (1984). An aggregate size distribution mode of 0.1 mm was used to estimate the threshold friction velocity, u_{*t}. This particle size, which is the smallest particle size characterized in the Cowherd model, was considered to be representative of the soils in the OBG area based on an analysis of soil sieve tests conducted at the site. Based on Figure 3-4 in Cowherd, the threshold surface friction velocity corresponding to the particle size mode is 25 cm/sec.

The threshold surface friction velocity describes the wind speed required at the soil surface to initiate entrainment of soil particles. The Cowherd PM_{10} emission rate equation requires input of the lowest wind speed, measured at a height of 7 m, which could initiate wind erosion. Frictional drag at the earth's surface causes a deceleration of the wind. This results in a wind speed profile that shows an increase in speed with increasing height. The wind speed profile in the lower atmosphere can be approximated by a logarithmic equation known as the logarithmic wind profile. This equation was used to convert the threshold surface friction velocity to the corresponding wind at 7 m. This conversion requires specification of the aerodynamic roughness of the surface over which the wind is flowing. For the OBG area at MAAP, an aerodynamic roughness of 0.03 m, which is representative of open flat terrain with grass and a few isolated obstacles (NOAA 1983) was used. Based on the logarithmic equation, the threshold friction velocity at 7 m was calculated to be 0.25 m/s.

The Cowherd PM_{10} emission equation also requires specification of the average wind speed in the area and the fraction of vegetative cover on the soil surface. The average wind speed which was obtained from the National Weather Service observations recorded at Jackson, TN is 3.7 m/sec. The fraction of soil covered by vegetation at the site was assumed to be 60%. This assumption was based on observations of ICF personnel who worked at the site.

The entrainment of soil particles via wind erosion will occur only when the wind speed is greater than or equal to the threshold wind speed. The Cowherd PM_{10} emission rate equation includes a weighting function which represents the periods when the wind can be expected to meet or exceed the threshold wind speed. This weighting function assumes that the wind speed distribution for a given site can be represented by a Rayleigh distribution. Using the function depicted by

Cowherd et al. (1984) in Figure 4-3, the value of the weighting function was estimated to be 1.91. The values for the input parameters used to calculate the PM_{10} emission factor are listed in Table O-1. Having specified the required input parameters, the emission factor for PM_{10} emissions was calculated from the following predictive equation developed from Gillette's (1981) field measurements of highly erodible soil:

$$E_{10w} = (0.036)(1-V)\left(\frac{u}{u_t}\right)^3 F(x)$$

where:

E_{10w} = annual average PM_{10} emission factor per unit area of contaminated surface ($g/m^2/hr$)

v = fraction of contaminated surface with vegetative cover (equals 0 for bare soil)

u = mean annual wind speed (m/sec)

u_t = threshold value of wind speed at an elevation of 7 m (m/sec)

x = $0.866(u_t/u)$ (dimensionless ratio)

F(x) = function related to the expected value of the wind speed (dimensionless)

The unit flux rate, E_{10w}, was calculated to be 3.27×10^{-2} g/m^2-hr. To obtain the annual chemical-specific PM_{10} emission rate due to wind erosion, R_{10}, the PM_{10} emission factor, E_{10W}, is multiplied by the mass fraction (in g/g) of the chemical measured in the soil using the following equation:

$$R_{10} = W \times E_{10W} \ (2.78 \times 10^{-4} \ hr/sec)$$

where:

R_{10} = emission flux of contaminant ($g/m^2/sec$)

E_{10W} = annual PM_{10} emission rate due to wind erosion ($g/m^2/hr$)

W = mass fraction of chemical in surface soils (g/g)

The mass fraction for each chemical used in the PM_{10} emission rate equation was obtained by converting the reported chemical concentrations in soil (in mg/kg) to a unitless value of g/g by using the appropriate conversion multipliers. It was assumed that the concentration in the PM_{10} particles was equal to the chemical concentrations measured in the bulk soil. Table O-2 lists these values as

TABLE O-1

INPUT PARAMETERS TO COWHERD (1984) WIND EROSION MODEL FOR SURFACES WITH AN "UNLIMITED RESERVOIR" OF ERODIBLE PARTICLES

V	=	60%
u	=	3.7 m/sec
u_t	=	0.25 m/sec
x	=	0.060
$\dot{F}(x)$	=	1.91

TABLE O-2

ESTIMATED EMISSION RATES FROM WIND EROSION OF SURFACE SOILS
IN THE OBG AREA AT MAAP

Chemical (a)	RME Concentration in Surface Soil (g/g)	Chemical-Specific Emission Rate (b) (g/m2-sec)
Explosives (c):		
HMX (HMX)	3.40E-04	3.09E-09
Nitrobenzene (NB)	4.30E-06	3.91E-11
RDX (RDX)	3.30E-03	3.00E-08
1,3,5-TNB (135TNB)	2.30E-06	2.09E-11
2,4,6-TNT (246TNT)	4.10E-03	3.73E-08
Inorganics (d):		
Arsenic (AS)	9.80E-06	8.92E-11
Chromium (CR)	3.50E-05	3.18E-10
Lead (PB)	9.30E-05	8.46E-10
Mercury (HG)	1.40E-06	1.27E-11
Silver (AG)	1.00E-07	9.10E-13
Zinc (ZN)	9.48E-05	8.63E-10

(a) USATHAMA chemical codes are listed in parentheses.
(b) Emission rates are calculated by multiplying the chemical concentration in soil (in g/g) by the unit emission rate of 9.10 x 10-6 g/m2-sec predicted using the Cowherd (1984) emissions model.
(c) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, and OBGB-4.
(d) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, OBGA-6, OBGB-4, OBGB-5, OBGC-4, OBGC-5, OBGD-3, and OBGD-4.

well as the R_{10} emission rates for the chemicals of potential concern. The predicted emission rates were used to estimate the chemical-specific ambient concentrations on- and off-site by linking them to the appropriate air dispersion model. On-site ambient air concentrations were predicted using a Box model as described in Section O.3. Off-site ambient air concentrations were predicted using the Fugitive Dust Model (FDM), which is described in Section O.4.

O.3 BOX MODEL

Conventional gaussian dispersion models such as the simple screening method developed by Turner (1970) or the more complex FDM dispersion model are not applicable for determining ambient concentrations at or near the source of emission. For this study, a box model was used to estimate the ambient air concentrations for on-site workers at the OBG.

The box model assumes steady and spatially uniform conditions of dispersion so that the emissions from an area source are uniformly distributed throughout a box defined by the area of the source and the mixing height. The model requires steady-state emission rates, a constant wind vector, and that the crosswind distance of the area source is large in comparison to the downwind distance to the receptor. To meet these requirements, all emission rates were calculated for steady state, the wind speed was chosen to be the annual average wind speed recorded at a nearby airport, and the receptor location was the site of the area source. The only condition left to determine was the height of the box. Box models used on an urban scale often use the height of the daytime mixing layer, approximately 500 m, as the height of the box. For that definition to be appropriate, a downstream fetch on the order of tens of kilometers is required.. For the purposes of this assessment (downstream fetch ≈ 115 m), the mean vertical displacement of emissions as a function of stability and downwind distance should provide a reasonable analogy to the mixing height used in the larger scale box models.

In this assessment, the box model was applied to two different areas. One box encompasses the area in the OBG where inorganic chemicals were detected. The other box encompasses the area in the OBG where explosive chemicals were detected. All other box model parameters for the two areas were assumed to be the same as they are both located in the same general area in the OBG. The box area for the inorganic chemicals was assumed to be 1400 m by 1100 m or approximately 1,540,000 m^2 in area. The box encompasses those sample locations where inorganics were detected in surface soils (OBGA-3, OBGA-4, OBGA-6, OBGB-4, OBGB-5, OBGC-4, OBGC-5, OBGD-3 and

OBGD-4). The box area for the explosive chemicals was assumed to be 400 m by 580 m, or approximately 232,000 m^2 in area. This box encompasses those sample locations where explosives were detected in surface soil samples (OBGA-3, OBGA-4, and OBGB-4). Refer to Figure O-1 for the soil boring locations.

The height of the box for each area was determined using the following equation presented in Pasquill (1975):

$$X = 6.25\ Z_0\ [(H/Z0)\ \ln\ (H/Z_0) - 1.58(H/Z_0) + 1.58))]$$

where:

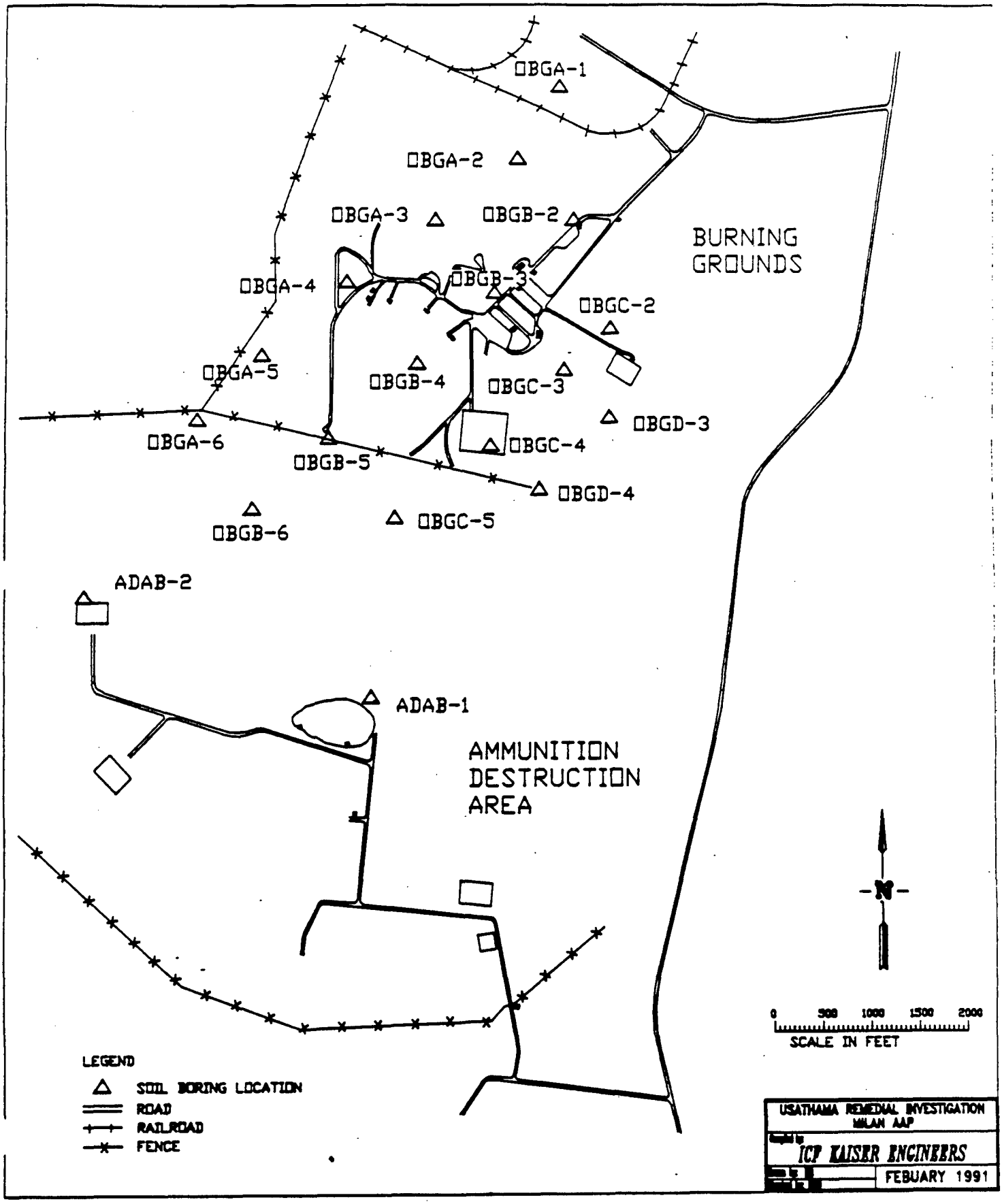
H	=	height of the box (m)
X	=	downwind distance [length of the box] (m)
Z	=	surface roughness height (m)

This expression is applicable for a D, or neutral stability class, and should provide an average estimate of ambient concentrations because the effect of a change in atmospheric stability would be to raise or lower the box height relative to the neutral stability. Changes in the box height will affect the ambient concentrations in the box since the volume available for diluting the emissions is changed. During daytime hours when workers could be exposed to windblown emissions, the atmospheric stability will be either neutral or unstable. An unstable atmosphere will have a larger box height relative to a neutral stability. Therefore, the use of a neutral stability for the box height calculation provides a conservative estimate of the potential inhalation impact for on-site workers.

Some modifications to the box model theory were made when estimating ambient air concentrations in the OBG. The box model described above assumes that the emissions occur at the upwind edge of the modelled box area and follow a straight-line trajectory until the emissions reach the final box height at the downwind edge of the box. However, to assume that all emissions will travel the entire length of the box and reach the final box height, H, may not be representative of the conditions. Particularly when the emission source is such as that at MAAP, where emissions occur over the entire modelled area and potential receptors (the on-site workers at OBG) are also located within the entire box area. Therefore, the box model was modified for such cases. This modified box model retains the assumption of a straight-line trajectory, but does not assume one constant downwind distance (X), box area, or height but instead calculates an average box height based on an iterative scheme. The downwind distance, X, is broken down into a smaller number of segments. An

Figure O-1

OBG and ADA Soil Boring Locations



iterative approach is then applied to the box model such that a new box height is calculated each time the box length is increased by adding another segment. All intermediate box heights are then averaged to estimate an average box height which is based on emissions originating at different areas of the box with varying downwind distances. The value for Z_0, the roughness height, was chosen to be 0.03 m which is representative of open flat terrain with grass and a few isolated obstacles (NOAA 1983). The downwind distance, X, for the inorganic chemicals box was chosen to be 1100 m. This downwind distance was divided into 10 segments (11 m each). The downwind distance, X, for the explosive chemicals box was chosen to be 400 m. This downwind distance was divided into 4 segments (100 m each).

Using the iterative technique described above, the average box height, H, was determined to be 19.4 m and 9.5 m, for the inorganic chemicals box and the explosive chemicals box, respectively.

The concentration on-site can be determined using the equation:

$$C_i = \frac{Q_{vi}}{HWU}$$

where:

C_i	=	The concentration on-site for the i^{th} contaminant (g/m^3)
Q_{vi}	=	Total emission rate of the i^{th} contaminant within the box (g/sec)
U	=	Average wind speed in the box (m/s)
W	=	Crosswind width of the area source (m)
H	=	Average height of the box (m)

The unit flux rate, 9.10 $x10^{-6}$ g/m^2-sec, calculated using the Cowherd (1984) emissions model described in Section O.2 above was input to the box model. An average wind speed value of 3.7 m/s which is based on the National Weather Service (NWS) data for Jackson, TN was used. For the inorganic chemicals box, a crosswind width of the area source of 1400 m and the average box height of 19.4 m was used. For the explosive chemicals box, a crosswind width of the area source of 580 m and the average box height of 9.5 m was used. Table O-3 summarizes the input parameters to the two box areas modeled and the resulting predicted box heights.

TABLE O-3

BOX MODEL INPUT PARAMETERS AND PREDICTED RESULTS

	Box 1[a]	Box 2[b]
Input Parameters:		
Total Box Area (m^2)	1,540,000	232,000
Downwind Distance (m)	1,100	400
Roughness Height (m)	0.03	0.03
Average Wind Speed (m/sec)	3.7	3.7
Emission Rate (g/m^2-sec)	9.10×10^{-6}	9.10×10^{-6}
Results:		
Average Box Height (m)	19.4	9.5

[a] Box model used to predict on-site air concentrations for inorganic chemicals detected at the OBG.

[b] Box model used to predict on-site air concentrations for explosive chemicals detected at the OBG.

The chemical-specific air concentrations estimated using the Box model are presented in Table O-4.

O.4 FUGITIVE DUST DISPERSION MODEL

The Fugitive Dust Model (FDM) is specifically designed for estimating downwind concentrations and deposition impacts from point, line or area fugitive dust sources (Winges 1990). The model was developed with the support of EPA Region X, which recommends that FDM be used for all fugitive dust sources such as surface mining activities, excavation, roadways, storage piles, and wind-blown emissions. As a result of these recommendations, this model was considered to be appropriate for estimating off-site annual air concentrations due to dust emissions from the OBG area at MAAP.

The FDM model is a steady-state Gaussian dispersion model which can be used to estimate ambient concentrations and deposition caused by fugitive dust sources. Concentrations and deposition can be computed for a maximum of 200 receptor locations. In this assessment, air concentrations only were predicted for a total of 25 receptors. These receptors encompassed a 400 m by 400 m area surrounding the closest off-site resident. Preliminary air dispersion modeling for 360° around the source to a downwind distance of 3.4 km indicated that the maximum off-site concentrations would occur in the area of these 25 receptors.

The model is designed to accept pre-processed (RAMMET) meteorological data, hourly meteorological data in card image format, or STability ARray (STAR) data. Five years of STAR data from Jackson, TN was used in this assessment. When the meteorological data are provided to the model in STAR format, the model will compute concentrations as 22.5 degree sector averages. The model smooths concentrations between adjacent sectors to eliminate large differences between adjacent receptors across sector boundaries.

Wind speed is used directly to determine the concentration, and is also used in combination with temperature and atmospheric stability to determine the values for deposition velocity if the deposition velocity is to be computed by the model. Wind direction is used to determine the location of the receptor with respect to the center point of each source in a coordinate system defined with the wind direction parallel to the x-axis. Atmospheric stability is also used to determine the values for the standard deviations of the horizontal and vertical plume dimension. The atmospheric stability for each

TABLE O-4

ESTIMATED ON-SITE AIR CONCENTRATIONS DUE TO WIND EROSION
OF CHEMICALS OF POTENTIAL CONCERN DETECTED IN THE SURFACE SOILS OF THE OBG

Chemical (a)	RME Concentration in Surface Soil (g/g) (b)	Concentration in Air (ug/m3) (c)
Explosives (d)		
HMX (HMX)	3.40E-04	3.54E-02
Nitrobenzene (NB)	4.30E-06	4.47E-04
RDX (RDX)	3.30E-03	3.43E-01
1,3,5-TNB (135TNB)	2.30E-06	2.39E-04
2,4,6-TNT (246TNT)	4.10E-03	4.26E-01
Inorganic Chemicals (e)		
Arsenic (AS)	9.80E-06	1.36E-03
Chromium (CR)	3.50E-05	4.86E-03
Lead (PB)	9.30E-05	1.29E-02
Mercury (HG)	1.40E-06	1.95E-04
Silver (AG)	1.00E-07	1.39E-05
Zinc (ZN)	9.48E-05	1.32E-02

(a) USATHAMA chemical codes are listed in parentheses.
(b) Surface soil samples are from the 0-1 ft. depth.
(c) Air concentrations are calculated by multiplying the soil concentration (in g/g) by the unit air concentration 1.39x10^2 ug/m3 and 1.04x10^2 ug/m3, for inorganic and explosive chemicals, respectively. These concentrations are based on the Cowherd (1984) wind erosion model and dispersion estimates are based on the Fugitive Dust Model (FDM). These are described in detail in Appendix O.
(d) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, and OBGB-4.
(e) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, OBGA-6, OBGB-4, OBGB-5, OBGC-4, OBGC-5, OBGD-3, and OBGD-4.

unit of meteorological data is specified as one of six possible stability classes, using the classification scheme of Turner (1970). The equations and coefficients for this classification are listed in the User's Guide for the Industrial Source Complex (ISC) Model (EPA 1987).

Because fugitive dust emissions are usually released at ground level, the model is generally insensitive to mixing height values since reflections off the mixing height are only significant at very far distances from the source or at elevated receptors. However, the model does consider such reflections when necessary.

Friction velocities are calculated internally in the FDM model using the methodology described in (McRae 1977). The algorithm requires a user specified roughness height, the wind speed and the reference height of the meteorological data. Each particle size class is treated separately by the model. The concentrations for different particle size classes are summed to develop a total suspended particulate concentration. For this assessment, only respirable particulate matter (aerodynamic diameter less than 10 μm) was considered. The particle sizes were divided into 5 classes: 10 μm, 8 μm, 6 μm, 4 μm and <2 μm. Based on these classifications, the average percentage of particles within each particle size range was determined. It was determined that soil borings OBGB-3, MI-080, CBG-1, and CLF-5 are representative of surface soils in the OBG area.[1] Therefore, the percentage of particles within each particle size range is based on an average of these four soil borings. The percentage of particles in each particle size class are presented in Table O-5.

Other input parameters required by FDM include a roughness length and a particle density. A roughness length of 0.03 m, representative of open flat terrain with grass and a few isolated obstacles (NOAA 1983), was used. A bulk particle density of 2.65 g/cm^3 was used, based on information obtained from site soil analyses.

Area sources are specified by the user with a center point, an x-dimension, and a y-dimension. The source areas modeled in this assessment are the same areas modeled using the Box model as described in Section O.3. The areas are as follows: a 1400 m x 1100 m area and a 400 m x 580 m area for the inorganic chemicals and explosive chemicals, respectively.

[1]Audry Orndorff, ICF/Kaiser Engineers; personal communcation, April 5, 1991.

TABLE O-5

PARTICLE SIZE DISTRIBUTION FOR PARTICLES LESS THAN 10 μm

Particle Size	Average Percent in Category[a]
<2 μm	0.55
4 μm	0.05
6 μm	0.10
8 μm	0.15
10 μm	0.15

[a] Based on an average of the percentage of particles within these categories for surface soil samples OBG B-3, MI-080, CBG-1, and CLF-5.

All major input parameters used in the model are summarized in Table O-6. Also presented in this table are the unit predicted air concentrations. The concentrations were then multiplied by the mass fraction (in g/g) of the concentrations of the chemicals of potential concern in the soil to estimate the chemical-specific concentrations in air for the nearest off-site receptor to the OBG area. These chemical-specific concentrations are summarized in Table O-7.

TABLE O-6

FUGITIVE DUST MODEL INPUT PARAMETERS AND PREDICTED RESULTS

	Run 1[a]	Run 2[b]
Input Parameters:		
Roughness Height (m)	0.03	0.03
Emission Rate (g/m^2-sec)	9.10×10^{-6}	9.10×10^{-6}
Source Dimensions (m x m)	1,400 m x 1,100 m	580 m x 400 m
Results:		
Unit Air Concentration at (0, 2400)	7.80 $\mu g/m^3$	1.12 $\mu g/m^3$

[a] Run to predict off-site air concentration at receptor point (0, 2400) due to dust emissions of inorganic chemicals detected at the OBG.

[b] Run to predict off-site air concentration at receptor point (0, 2400) due to dust emissions of explosive chemicals detected at the OBG.

TABLE O-7

ESTIMATED OFF-SITE AIR CONCENTRATIONS DUE TO WIND EROSION
OF CHEMICALS OF POTENTIAL CONCERN DETECTED IN THE SURFACE SOILS OF THE OBG (a)

Chemical (b)	RME Concentration in Surface Soil (g/g) (c)	Concentration in Air (ug/m3) (d)
Explosives (e)		
HMX (HMX)	3.40E-04	3.81E-04
Nitrobenzene (NB)	4.30E-06	4.82E-06
RDX (RDX)	3.30E-03	3.70E-03
1,3,5-TNB (135TNB)	2.30E-06	2.58E-06
2,4,6-TNT (246TNT)	4.10E-03	4.59E-03
Inorganic Chemicals (f)		
Arsenic (AS)	9.80E-06	7.64E-05
Chromium (CR)	3.50E-05	2.73E-04
Lead (PB)	9.30E-05	7.25E-04
Mercury (HG)	1.40E-06	1.09E-05
Silver (AG)	1.00E-07	7.80E-07
Zinc (ZN)	9.48E-05	7.39E-04

(a) The nearest off-site receptor is located approximately 2.4 km east of the OBG area at MAAP (FDM model receptor points [2400 m, 0 m]).
(b) USATHAMA chemical codes are listed in parentheses.
(c) Surface soil samples are from the 0-1 ft. depth.
(d) Air concentrations are calculated by multiplying the soil concentration (in g/g) by the unit air concentration 7.80 ug/m3 and 1.12 ug/m3, for inorganic and explosive chemicals, respectively. These concentrations are based on the Cowherd (1984) wind erosion model and dispersion estimates are based on the Fugitive Dust Model (FDM). These are described in detail in Appendix O.
(e) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, and OBGB-4.
(f) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, OBGA-6, OBGB-4, OBGB-5, OBGC-4, OBGC-5, OBGD-3, and OBGD-4.

O.5 REFERENCES

COWHERD, C., MULESKI, G.E., ENGLEHART, P.J. and GILLETTE, D.A. 1984. Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Site. Midwest Research Inst., Kansas City, MO. PB85-192219

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Industrial Source Complex (ISC) Dispersion Model User's Guide -- Second Edition (Revised). Volume I. Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-450/4-88-002a.

GILLETTE, D.A. 1981. Production of dust that may be carried great distances. In Desert Dust: Origin, Characteristics, and Effect on Man, edited by Troy Pewe. Geological Society of American Special Paper 186, pp. 11-26.

MCRAE, G.J. 1977. Computer Program for calculation of the friction velocity, Environmental Quality Laboratory, 206-40, California Institute of Technology, Pasadena, CA 91125.

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION (NOAA). 1983. Preparing Meteorological Data for Use in Routine Dispersion Calculations -- Workgroup Summary Report. Air Resources Laboratory, Rockville, Maryland. August 1983.

PASQUILL, F. 1975. The dispersion of material in the atmospheric boundary layer - the basis for generalization. In: Lectures on Air Pollution and Environmental Analysis. Boston, Mass: American Meteo. Soc.

TURNER, D.B. 1970. Workbook of Atmospheric Dispersion Estimates. Environmental Protection Agency. Office of Air Programs, Research Triangle Park, N.C.

WINGES, K.D. 1990. User's Guide for the Fugitive Dust Model (FDM) (Revised). Volume I: User's Instructions. Prepared for Region 10, U.S. Environmental Protection Agency, Seattle, Washington.

APPENDIX P

HUMAN HEALTH TOXICITY PROFILES

ACETONE

Acetone is absorbed in humans and animals following oral or inhalation exposure (EPA 1984). Approximately 75 percent of inhaled vapor is absorbed by the pulmonary route (Kagan 1924). Acute exposure to acetone vapors of 500 ppm produce irritation of the mucosal membranes in humans (EPA 1984, Nelson et al. 1943). Prolonged or repeated dermal contact may defat the skin and produce dermatitis (Krasavage et al. 1981). Rats acutely exposed to acetone vapors showed behavioral changes as demonstrated by an inability to pole climb following stimulation (Goldberg et al. 1964). In rats, slight increases in organ weights, decreases in body weights, and nephrotoxicity have been observed following long-term oral exposure to acetone (EPA 1986). Humans chronically exposed to atmospheric concentrations in excess of 10,000 ppm are likely to experience central nervous system depression and narcotic effects (Krasavage et al. 1981).

EPA (1991) derived an oral reference dose (RfD) for acetone of 0.1 mg/kg/day based on a study sponsored by the EPA Office of Solid Waste (EPA 1986) in which increased liver and kidney weights and nephrotoxicity were observed in rats exposed orally to acetone; an uncertainty factor of 1,000 was used to derive the RfD. EPA (1990) derived a subchronic oral RfD of 1 mg/kg/day using an uncertainty factor of 100, based on the same study and effect of concern.

ALUMINUM

Little is known about the absorption of aluminum following oral exposure, although there are reports that this inorganic metal is absorbed from the gastrointestinal tract to some extent (NAS 1982). Aluminum has low acute toxicity following oral exposure. Oral LD_{50}s in several animal species range from 380 to 780 mg/kg (EPA 1985). Neurofibrillar degeneration has been observed in experimental animals given subcutaneous injections of aluminum (Goyer 1986). Intratracheal and intraperitoneal administration of aluminum compounds have been associated in experimental animals with pulmonary fibrosis and fibrotic peritonitis, respectively (NAS 1982). Evidence to date suggests that aluminum is not teratogenic or fetotoxic; however, rats exposed by gavage to 2.5 mg/kg aluminum for 6 months had reduced sperm counts and reduced sperm motility (EPA 1985). Studies to evaluate the potential of aluminum or its salts to induce mutagenic or carcinogenic effects have yielded negative results

APPENDIX P

HUMAN HEALTH TOXICITY PROFILES

(EPA 1985). In humans, pulmonary fibrosis has been observed following inhalation of aluminum fumes. Aluminum has been presumed to be a potential etiological factor in two neurological disorders: Alzheimer's disease and chronic renal failure accompanied by senile dementia (Goyer 1986). However, the importance of aluminum in these disorders had not been fully established. Because there are inadequate dose-response data from which to estimate an acceptable daily intake level for ionic aluminum. no health-based criteria have been established by EPA.

ARSENIC

Both inorganic and organic forms of arsenic are readily absorbed via the oral and inhalation routes. Soluble forms are more readily absorbed than insoluble forms (EPA 1984). Approximately 95% of soluble inorganic arsenic administered to rats is absorbed from the gastrointestinal tract (Coulson et al. 1935, Ray-Bettley and O'Shea 1975). Approximately 70%-80% of arsenic deposited in the respiratory tract of humans has been shown to be absorbed (Holland et al. 1959). Dermal absorption is not significant (EPA 1984). Acute exposure of humans to metallic arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy (EPA 1984). Chronic exposure of humans to this metal can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular damage (EPA 1984, Tseng 1977). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (EPA 1984). Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma (EPA 1984). Ingestion of arsenic has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer (Tseng 1977, Tseng et al. 1968, Chen et al. 1986).

EPA has classified arsenic in Group A—Human Carcinogen—and has developed inhalation (EPA 1991) and oral (EPA 1988) slope factors of 50 $(mg/kg/day)^{-1}$ and 2.0 $(mg/kg/day)^{-1}$, respectively. The inhalation potency factor is the geometric mean value of potency factors derived from four occupational exposure studies on two different exposure populations (EPA 1984). The oral cancer potency factor was based on an epidemiological study in Taiwan which indicated an increased incidence of skin cancer in individuals exposed to arsenic in drinking water (Tseng 1977). A risk assessment for noncarcinogenic effects of arsenic is currently under review by EPA (1991). An oral reference dose (RfD) of $1x10^{-3}$ mg/kg/day was calculated for arsenic based on the same oral epidemiological study (Tseng 1977) which also showed greater incidence of keratosis and

hyperpigmentation in humans (EPA 1990). An uncertainty factor of 1 was used to derive the oral RfD. This RfD is presently being reconsidered by the RfD workgroup.

BARIUM

Adverse effects in humans following oral exposure to soluble barium compounds include gastroenteritis, muscular paralysis, hypertension, ventricular fibrillation, and central nervous system damage (EPA 1984). Inhalation of barium sulfate or barium carbonate in occupationally exposed workers has been associated with baritosis, a benign pneumoconiosis (Goyer 1986). Human epidemiologic studies have shown that chronic ingestion of drinking water containing high levels of barium induces high blood pressure that results in a prevalance of hypertension, stroke, heart and renal disease (Brenniman and Levy 1984, Wones et al. 1990). Chronic oral exposure of experimental animals to barium in drinking water also increases blood pressure (EPA 1984, Perry et al. 1983). Inhalation of barium carbonate dust by experimental animals has been associated with reduced sperm count, increased fetal mortality, atresia of the ovarian follicles, decreased body weight, and alterations in liver function (EPA 1984, Tarasenko et al. 1977).

EPA (1991) derived an oral reference dose (RfD) based on two human epidemiologic studies which did not observe any adverse effects following consumption of drinking water containing barium (Brenniman and Levy 1984, Wones et al. 1990). Although no LOAEL was identified, the effect of concern was high blood pressure. Using a NOAEL of 0.21 mg/kg/day and an uncertainty factor of 3, an oral RfD of $7x10^{-2}$ mg/kg/day was calculated. A subchronic RfD of $5x10^{-2}$ mg/kg/day has been established by EPA (1990) based on increased blood pressure in rats chronically exposed to 5.1 mg barium/kg/day in their drinking water (Perry et al. 1983). EPA (1990) has also developed a chronic and subchronic inhalation RfD of $1.0x10^{-4}$ mg/kg/day and $1.0x10^{-3}$ mg/kg/day for barium based on a study by Tarasenko et al. (1977). In this study rats were exposed to barium carbonate dust at airborne concentrations of up to 5.2 mg/m^3 for 4-6 months. Adverse effects noted at this concentration included decreased body weight, alterations in liver function, and increased fetal mortality. Uncertainty factors of 1,000 and 100 were used in developing the RfDs.

BROMODICHLOROMETHANE

Bromodichloromethane, also known as dichlorobromomethane is readily absorbed following oral exposure (EPA 1984, NTP 1986). Compound-related cytomegaly and tubular cell hyperplasia of the

kidney and fatty metamorphosis of the liver have been observed in rats following chronic gavage administration (NTP 1986). Some fetal anomalies were observed in the offspring of mice exposed to very high levels of bromodichloromethane during gestation (EPA 1984). This chemical tested positive for mutagenicity in the Salmonella/microsome assay and in the L5178Y mouse lymphoma test (NTP 1986). Mice chronically administered bromodichloromethane by gavage developed an increased incidence of liver tumors (NTP 1986).

EPA (1991) has classified bromodichloromethane as a B2 agent (probable human carcinogen) indicating there is sufficient evidence of carcinogenicity in animals but inadequate evidence in humans. EPA (1991) has established an oral cancer potency factor of 0.13 $(mg/kg/day)^{-1}$ based on an increased incidence of liver tumors in mice (NTP 1986). EPA (1991) has derived an oral reference dose (RfD) for bromodichloromethane of $2x10^{-2}$ mg/kg/day based on a chronic gavage study in which renal cytomegaly was observed in mice (NTP 1986). An uncertainty factor of 1,000 was applied to a LOAEL of 17.9 mg/kg/day to derived the RfD.

BROMOFORM

Bromoform is rapidly absorbed following oral administration, with gastrointestinal absorption estimated to be 60 to 90% complete (Mink et al. 1986). Low-level inhalation exposure of humans to bromoform results in irritation, lacrimation and reddening of the face (Sax and Lewis 1989). Bromoform, like other halogenated hydrocarbrons, can induce central nervous system depression following both oral and inhalation exposures (ATSDR 1989). In experimental animals the target organs appear to be the CNS, liver and kidney following acute exposure (Dykan 1962, 1964, Bowman et al. 1978). Subchronic oral administration of rats resulted in liver and thyroid effects characterized by fatty liver infiltration and vacuolization, and a reduction in the follicular size (Chu et al. 1982, NTP 1980). In animals, chronic exposure produced nonneoplastic liver lesions manifested as fatty change, active chronic inflammation, and necrosis (NTP 1988). Increased incidences of uncommon tumors of the large intestine have been induced by chronic oral bromoform exposure (NTP 1988).

Bromoform is classified in B2 (EPA 1991). EPA (1991) has calculated an oral cancer potency factor of $7.9x10^{-3}$ $(mg/kg/day)^{-1}$ based on the development of adenomatous polyps or adenocarcinomas in the large intestine of rats chronically administered bromoform by gavage (NTP 1988). EPA (1991) has derived an oral reference dose (RfD) of $2x10^{-2}$ mg/kg/day for bromoform based on a subchronic oral bioassay conducted in rats (NTP 1980). A no-observed-adverse effect level (NOAEL) of 25 mg/kg/day

for hepatic lesions was identified, to which an uncertainty factor of 1,000 was applied to derive the RfD.

CADMIUM

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (EPA 1985a). Pulmonary absorption of cadmium in humans is reported to range from 10% to 50% (CDHS 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (EPA 1985a,b). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a renal cortex concentration of cadmium of 200 μg/g (EPA 1985b). Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lung, kidney, and prostate (EPA 1985b, Thun et al. 1985). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (EPA 1985b).

EPA (1991) classified cadmium as a Group B1 agent (Probable Human Carcinogen) by inhalation. This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies. EPA (1991) derived an inhalation cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ for cadmium based on epidemiologic studies in which respiratory tract tumors were observed (Thun et al. 1985, EPA 1985b). Using renal toxicity as an endpoint, and a safety factor of 10, EPA (1991) has derived two separate oral reference doses (RfD). The RfD associated with oral exposure to drinking water is $5x10^{-4}$ mg/kg/day, and is based upon the lowest-observed-adverse-effect level (LOAEL) of 0.005 mg/kg in humans (EPA 1985a, Friberg et al. 1974). The RfD associated with exposure to cadmium in food is $1x10^{-3}$ mg/kg/day.

CALCIUM

Calcium is an essential nutrient which comprises a major portion (90%) of the bone and is necessary for the functional integrity of the nerve and muscle fibers where it influences the excitability and release of neurotransmitters (Haynes and Murad 1985). The recommended daily allowance of calcium is 800 mg/day for adults. Approximately thirty percent of ingested calcium is absorbed primarily in the proximal segments of the small intestine (Haynes and Murad 1985). Prolonged deficiency may result

in osteoporosis and in newborn children, tetany (twitches and spasms) is prevalent. Calcium is relatively non-toxic when administered orally. Calcium salts may have toxic effects depending on the toxicity of the anion constituent. Examples of potentially toxic calcium salts include calcium chloride, flouride, bromide, and phosphate. Diets that are high in calcium have produced symptoms of zinc deficiency in rats, chickens and pigs after prolonged feeding (Hedsted 1957). Peach (1975) indicated that calcium intakes in excess of 1,000 mg/L when coupled with high vitamin D intake raise blood levels of calcium and can depress serum magnesium levels following prolonged periods (NRC 1980). Kidney stones in humans have been associated with high calcium intakes. Hypercalciemia, a pathological condition of retaining excess calcium in the body, results in increased nerve and muscle excitation. This is manifested clinically by muscle weakness, lethargy, and eventually coma. Kidney and renal dysfunction are also associated with excess calcium levels, with prominent pathological changes in the collecting ducts and distal tubules (Haynes and Murad 1985). There is no evidence that calcium is mutagenic, carcinogenic or teratogenic. No health based-criteria have been established by EPA.

CARBON DISULFIDE

Carbon disulfide is absorbed largely through the lungs but toxic quantities can also be absorbed through the skin. Carbon disulfide can be lethal in humans exposed to high oral doses or concentrated vapors. The nonlethal acute effect of carbon disulfide is narcosis (Sax 1984). Neurotoxicity is the primary effect of carbon disulfide exposure and manifests as neurophysiological and behavioral changes in the structure and function of the peripheral nervous system (ATSDR 1990). Adverse neurological effects of human exposure to prolonged, high levels of carbon disulfide include organic brain damage, peripheral nervous system decrements, neurobehavioral dysfunction, and ocular and auditory effects (Andrews and Snyder 1986). Symptoms include nervousness, irritability, indigestion, excessive fatigue, loss of appetite and headaches (ACGIH 1986). Repeated exposure to carbon disulfide may also cause cardiovascular and gastrointestinal effects in humans (ATSDR 1990). Longer-term exposure of animals to vapor concentrations of carbon disulfide include inreased serum lipids, histopathological changes in the myocardium, visual impairment, hindlimb paralysis, and lethargy (ATSDR 1990). Developmental effects in rats exposed in utero include behavioral and learning deficits; fetal malformations were evident at vapor concentrations which elicited maternal toxicity (Tabacova et al. 1983). Carbon disulfide has also been reported to increase the incidence of fetal resorption in rabbits exposed to 25 mg/kg/day carbon disulfide in water (Price et al. 1984).

Inhalation exposure of rabbits to concentrations of 11 mg/kg/day did not induce fetal toxicity or malformations (Hardin et al. 1981).

EPA (1991) derived an oral RfD for carbon disulfide of 0.1 mg/kg/day based on a study in which no fetal toxicity or malformations were observed in rabbits following inhalation exposure to 11 mg/kg/day carbon disulfide (Hardin et al. 1981). An uncertainty factor of 100 was used to develop the RfD. EPA (1990) established an inhalation RfC of $1x10^{-2}$ mg/m^3 for carbon disulfide based upon fetotoxicity and developmental effects in rabbits exposed via inhalation (Hardin et al. 1981).

CHLOROFORM

Chloroform, a trihalomethane, is rapidly absorbed through the respiratory and gastrointestinal tracts in humans and experimental animals; dermal absorption from contact of the skin with liquid chloroform can also occur (EPA 1985). In humans, acute exposures to chloroform may result in depression of the central nervous system, hepatic and renal damage, and death caused by ventricular fibrillation following an acute ingested dose of 10 ml (EPA 1984). Acute exposure to chloroform may also cause irritation to the skin, eyes, and gastrointestinal tract (EPA 1984, 1985). In experimental animals, chronic exposure may lead to fatty cyst formation in the liver (Heywood et al. 1979), renal and cardiac effects, and central nervous system depression (EPA 1985). Chloroform has been reported to induce renal epithelial tumors in rats (Jorgenson et al. 1985) and hepatocellular carcinomas in mice (NCI 1976). Suggestive evidence from human epidemiological studies indicates that long-term exposure to chloroform and other trihalomethanes in contaminated water supplies may be associated with an increased incidence of bladder tumors (EPA 1985). Chloroform is embryotoxic in pregnant rats and has retarded fetal development and increased the incidences of fetal resorption, absence of tail, imperforate anus, missing ribs and delayed ossification of sternebrae (Schwetz et al. 1974).

Chloroform has been classified by EPA (1991) as a Group B2 Carcinogen (Probable Human Carcinogen). EPA (1991) developed an oral cancer potency factor for chloroform of $6.1x10^{-3}$ $(mg/kg/day)^{-1}$ based on a study in which kidney tumors were observed in rats exposed to chloroform in drinking water (Jorgenson et al. 1985). An inhalation cancer potency factor of $8.1x10^{-2}$ $(mg/kg/day)^{-1}$ has been developed by EPA (1990a) based on an NCI (1976) bioassay in which liver tumors were observed in mice. EPA also derived an oral reference dose (RfD) of 0.01 mg/kg/day for both chronic (EPA 1991) and subchronic (EPA 1990) exposure to chloroform based on a chronic bioassay in dogs

in which liver effects were observed at 12.9 mg/kg/day (Heywood et al. 1979); an uncertainty factor of 1,000 was used to derive the RfD for both exposure levels.

CHROMIUM

Chromium exists in two states, as chromium (III) and as chromium (VI). Following oral exposure, absorption of chromium (III) is low while absorption of chromium (VI) is high (EPA 1987). Chromium is an essential micronutrient and is not toxic in trace quantities (EPA 1980). High levels of soluble chromium (VI) and chromium (III) can produce kidney and liver damage following acute oral exposure. CNS effects including hypoactivity have been reported in rats when exposed to acute and subchronic levels of chromium VI in drinking water (Diaz-Mayans et al. 1986). Chronic inhalation exposure may cause damage to the respiratory tract (EPA 1984). Further, epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (Mancuso 1975, EPA 1984). Inhalation of chromium (III) or ingestion of chromium (VI) or (III) has not been associated with carcinogenicity in humans or experimental animals (EPA 1984). Certain chromium salts have been shown to be teratogenic and embryotoxic in mice and hamsters following intravenous or intraperitoneal injection (EPA 1984).

EPA has classified inhaled chromium (VI) in Group A--Probable Human Carcinogen by the inhalation route (EPA 1991). Inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity (EPA 1991). EPA (1991) developed an inhalation unit risk of 1.2x10-2 $(ug/m^3)^{-1}$ chromium (VI) based on an increased incidence of lung cancer in workers exposed to chromium over a 6 year period, and followed for approximately 40 years (Mancuso 1975). This is equivalent to a slope factor of 42 $(mg/kg/day)^{-1}$, assuming a 70 kilogram individual inhales 20 m^3/day. EPA (1991) derived a chronic oral reference dose (RfD) of $5.0x10^{-3}$ mg/kg/day for chromium (VI) based on a study by MacKenzie et al. (1958) in which no adverse effects were observed in rats exposed to 2.4 mg chromium (VI)/kg/day in drinking water for 1 year. A safety factor of 500 was used to derive the RfD. EPA (1990) calculated a subchronic RfD for chromium VI of $2x10^{-2}$ mg/kg/day based on the same study, effect of concern and using a safety factor of 100. EPA (1991) developed an oral RfD of 1 mg/kg/day for chromium (III) based on a study in which rats were exposed to chromic oxide baked in bread; no effects due to chromic oxide treatment were observed at any dose level (Ivankovic and Preussman 1975), however hepatotoxicity was the effect of concern. EPA (1990) established a subchronic RfD of 10 mg/kg/day for chromium III based on the same study and

endpoint. Safety factors of 1,000 and 100 were used to calculate the chronic and subchronic oral RfDs, respectively. EPA (1990) has derived an inhalation RfC of $2x10^{-6}$ mg/m^3 for both chromium III and chromium VI based on nasal mucosa atrophy in humans occupationally exposed to chromic acid (Lindberg and Hedenstierna 1983). This value is equivalent to $5.7x10^{-7}$ mg/kg/day, assuming a 70 kilogram individual inhales 20 m^3/day. An uncertainty factor of 300 was used to calculate the inhalation RfC.

COBALT

Cobalt is an essential trace element in human nutrition. Cobalt is generally well absorbed following ingestion. Acute ingestion of large doses by humans produces gastrointestinal disturbances (vomiting, diarrhea), and a sensation of warmth. Signs of acute poisoning in animals fed cobalt salts consist of diarrhea, loss of appetite, paralysis of hind legs, cutaneous vasodilation, and reduced body temperature prior to death. Large doses produced anuria, while smaller doses resulted in albuminuria (Stokinger 1981). In animals, subchronic oral exposures result in disturbed conditioned reflexes and alterations in hematopoiesis (NRC 1977). In humans, chronic oral exposure to cobalt in high doses can cause goiter, decreased thyroid function, increased heart and respiratory rates and blood lipid changes (Hammond and Beliles 1980). Chronic exposure to cobalt dust has been reported to produce respiratory disease in workers (Stokinger 1981). Cobalt salts included in a beer formulation at concentrations 1.2 to 1.5 mg/liter were reported to be responsible for a number of deaths due to congestive heart failure (NRC 1977). Cobalt administered to laboratory rodents produced adverse teratogenic effects including craniofacial developmental abnormalities in mice (Leonard et al. 1984) and decreased body weight in rats (Shepard 1986). Cobalt has been reported to cause sarcomas at the site of injection in rats (Gilman 1962, Heath 1960); however, the results of carcinogenesis studies performed by other routes of exposure have been negative. EPA has not classified cobalt on the basis of its carcinogenicity and no health based standards have been derived.

COPPER

Copper is an essential element. A daily copper intake of 2 mg is considered to be adequate for normal health and nutrition; the minimum daily requirement is 10 μg/kg (EPA 1985). In humans, absorption of copper following oral exposure is approximately 60% and is influenced by competition with other metals and the level of dietary protein and ascorbic acid in both humans and animals (EPA 1984). Copper is absorbed following inhalation exposures, although quantitative data on the extent of

absorption are unavailable (EPA 1984). Adverse effects in humans resulting from acute exposure to copper at concentrations that exceed these recommended levels by ingestion include salivation, gastrointestinal irritation, nausea, vomiting, hemorrhagic gastritis, and diarrhea (ACGIH 1986). Dermal or ocular exposure of humans to copper salts can produce irritation (ACGIH 1986). Acute inhalation of dusts or mists of copper salts by humans may produce irritation of the mucous membranes and pharynx, ulceration of the nasal septum, and metal fume fever. The latter condition is characterized by chills, fever, headache, and muscle pain. Limited data are available on the chronic toxicity of copper; however, chronic over-exposure to copper by humans has been associated with anemia (ACGIH 1986) and local gastrointestinal irritation (EPA 1987). Results of several animal bioassays suggest that copper compounds are not carcinogenic by oral administration; however, some copper compounds can induce injection-site tumors in mice (EPA 1985).

EPA (1990) has reported the drinking water standard of 1.3 mg/liter as an oral reference dose (RfD) for both chronic and subchronic exposures based on local gastrointestinal irritation (EPA 1987). Assuming a 70-kg adult ingests 2 liters of water per day, this concentration is equivalent to a dose of $3.7x10^{-2}$ mg/kg/day. However, EPA (1987) concluded toxicity data were inadequate for the calculation of a reference dose (RfD) for copper. An uncertainty factor of 1 was used to derive the oral RfD.

DIBROMOCHLOROMETHANE

Dibromochloromethane (DBCM) also known as chlorodibromomethane, is absorbed 60 to 90% in rats and mice following oral exposure (Mink et al. 1986). Human absorption data and animal absorption data through other routes is not available. Acute oral and inhalation exposure to high doses of DBCM can cause depression of the central nervous system, and injury to the liver and kidney (ATSDR 1989). In animals, acute effects on the central nervous system include ataxia, sedation, and labored breathing (Balster and Borzelleca 1982, Bowman et al. 1978, NTP 1985). Acute exposure (14 days) of mice to 125-250 mg/kg/day to DBCM resulted in a decrease in several indices of humoral and cell mediated immunity (Munson et al. 1982). Most studies of the toxicity of DBCM in rats and mice indicate that the liver is the target tissue. Although hepatic effects are not severe, they are characterized by increased vacuolization, fat accumulation, increased liver weight, and altered serum enzyme levels (Condie et al. 1983, Munson et al. 1982, NTP 1985). There also appear to be species and sex sensitivites for hepatic effects. Mice show a greater sensitivity to DBCM than rats, and males tend to be more sensitive than females (Condie et al. 1983, NTP 1985). These sensitivites may be related to differences in the metabolism of this compound by the respective species and sex. Renal

effects characterized as tubular degeneration and mineralization have been reported following longer-term exposure regimens (NTP 1985). Studies of the reproductive effects of DBCM in animals indicate that oral exposure does not result in significant damage to male or female reproductive organs (NTP 1985). Likewise, animal studies of the developmental effects of oral exposure to DBCM do not result in fetotoxicity or teratogenicity (Ruddick et al. 1983).

EPA (1991) has classified DBCM as a B2 agent and has reported an oral cancer potency factor of 0.084 based on a chronic (102 week) study investigating hepatic effects in mice (NTP 1985). EPA (1991) derived an oral reference dose (RfD) of 0.02 mg/kg/day for DBCM (chlorodibromomethane) based on a chronic study which observed hepatic lesions. An uncertainty factor of 1,000 was used to develop the RfD.

DIETHYLPHTHALATE

Diethylphthalate (DEP) is absorbed following ingestion and inhalation exposures. Its acute toxicity for laboratory animals by most routes of administration is very low (NIOSH 1986). Exposure of humans to the heated vapor may cause respiratory irritation (ACGIH 1986). No specific lesions were observed in subchronic or chronic feeding studies of DEP to rats and dogs. However, decreased consumption of food and increased relative organ weights were observed in some of the animals (EPA 1980, EPA 1986, Brown et al. 1978). Workers chronically exposed to DEP experienced pain, numbness, and spasms in the upper and lower extremities (ACGIH 1986). Reduced fetal weight, resorptions and dose-related musculoskeletal abnormalities were observed among fetuses from rats exposed to DEP intraperitoneally during gestation (EPA 1980). Studies indicate that DEP is mutagenic in bacterial test systems (EPA 1986, Seed 1982). Currently, no information is available on the carcinogenic potential of DEP in humans or animals.

EPA (1991) calculated an oral reference dose (RfD) of $8x10^{-1}$ mg/kg/day based on a subchronic rat study in which decreased growth rate, food consumption, and altered organ weights were the observed effects (Brown et al. 1978). The oral RfD was derived using a no-observed-adverse-effect level (NOAEL) of 750 mg/kg/day and an uncertainty factor of 1,000.

DINITROBENZENES

Absorption of the dinitrobenzene (DNB) isomers (1,2-DNB, 1,3-DNB, and 1,4-DNB) have not been well characterized, however ready absorption through the skin is a major factor in its toxicity (ACGIH 1986). Occupational exposures to the DNB isomers have been associated with methemoglobinemia and respiratory tract irritation. Prolonged exposures of humans to dinitrobenzene may result in anemia, liver damage and cyanosis (Beard and Noe 1981). In animals, subchronic oral exposures have resulted in retarded growth, decreased hemoglobin concentrations, splenic enlargement and hemosiderin deposits. Testicular atrophy and decreased spermatogenesis have also been observed in male rats following oral exposures (Cody et al. 1981).

EPA (1991) has developed an oral reference dose (RfD) of 1.0×10^{-4} mg/kg/day for 1,3-DNB based on a subchronic drinking water study in rats. This study identified a lowest-observed-effect-level (LOEL) of 8 ppm for increased splenic weight and a no-observed-effect-level (NOAEL) of 3 ppm (0.40 mg/kg/day) (Cody et al. 1981). The RfD was calculated using the NOAEL and an uncertainty factor of 3,000.

2,4-DINITROTOLUENE

2,4-Dinitrotoluene (2,4-DNT) is absorbed by inhalation of its vapor and via the gastrointestinal tract (EPA 1980). The most important acute toxic effect elicited by 2,4-DNT is the induction of methemoglobinemia followed by cyanosis. In laboratory mammal species oral LD_{50} values for 2,4-DNT have been reported to range between 268 to 650 mg/kg for rats, and from 1,250 to 1,954 mg/kg for mice (Etnier 1987). The blood, liver, and neuromuscular systems are the primary target organs for 2,4-DNT toxicity. In humans, symptoms reportedly caused by exposure to 2,4-DNT include vertigo, fatigue, nausea, dyspnea, drowsiness, tremors, paralysis, unconsciousness, chest pain, and heart palpitations (EPA 1980). Nonlethal acute responses to 2,4-DNT exposure in experimental animals include CNS depression, respiratory depression, and ataxia (Rickert et al. 1984). In animals, subchronic oral administration has resulted in muscular incoordination, methemoglobinemia, anemia, hematopoiesis in the spleen, hepatic proliferative lesions, gliosis and demyelination in the brain and atrophy with aspermatogenesis in the testes (Ellis et al. 1976, 1979). Chronic exposure may produce liver damage, jaundice and reversible anemia due to blood damage (Linch 1974, Key et al. 1977, Proctor and Hughes 1978). 2,4-DNT is a dermal irritant and allergen (Sax and Lewis 1989). Data concerning the mutagenicity of this compound is limited and conflicting. It was shown to cause

reverse and forward mutations in several strains of Salmonella typhimurium (Couch et al. 1981, Tokiwa et al. 1981). DNA repair was shown to occur in an in vivo male rat hepatocyte assay (Mirsalis and Butterworth 1982), but negative results were obtained in vitro assays using rat hepatocytes (Bermudez et al. 1979) and spermatocytes (Working and Butterworth 1984). 2,4-DNT induces mammary tumors, and hepatocellular carcinomas in rats and tumors of the kidney in mice following chronic oral administration (Ellis et al. 1979). NCI (1978) reported that dietary administration of 2,4-DNT to rats induced benign tumors of the skin and subcutaneous tissue and mammary fibroadenomas, and that mice developed alveolar/bronciolar adenomas, and tumors of the pituitary.

2,4-DNT has been classified by EPA as a B2 agent--Probable Human Carcinogen (EPA 1991). EPA (1991) has developed an oral cancer potency factor of $6.8x10^{-1}$ $(mg/kg/day)^{-1}$ based on an increased incidence of benign and malignant tumors in the liver and mammary gland in rats and malignant renal tumors in male mice administered 2,4-DNT (98% 2,4-DNT and 2% 2,6-DNT) in the diet for two years (Ellis et al. 1979).

2,6- DINITROTOLUENE

2,6-Dinitrotoluene (2,6-DNT) is rapidly absorbed from the gastrointestinal tract; inhalation and skin absorption of the DNT isomers may also occur (Etnier 1987). The principal route of excretion of 2,6-DNT and its metabolites is via the urine, with excretion virtually complete after 24 hours (Etnier 1987). In laboratory mammal species (potential surrogates for wildlife species), oral LD_{50} values for 2,6-DNT range between 177 and 795 mg/kg for rats, and between 621 and 1,000 mg/kg for mice (Etnier 1987). 2,6-DNT is a mild skin irritant in rabbits and a mild sensitizing agent in guinea pigs (Lee et al. 1975). The blood, liver, and neuromuscular systems are the primary target organs for DNT toxicity in both humans and experimental animals. Subchronic and chronic exposures of workers to 2,4- and 2,6-DNT in munitions plants have resulted in symptoms of cyanosis, dizziness, headache, dyspnea, and methemoglobinemia (Hamblin 1963, von Oettingen 1941, Etnier 1987). Long term occupational exposures have also been correlated with an increase in ischemic heart disease (Levine et al. 1986). The effects of 2,6-DNT following subchronic oral exposure in dogs, rats and mice include bile duct hyperplasia, degenerative changes in the spleen and liver, decreased RBC counts, testicular atrophy and aspermatogenesis (Lee et al. 1976). Leonard et al. (1987) reported significant reductions in body weight gain, increased liver weight and elevated serum alanine transferase and gamma-glutamyl transferase in rats administered 2,6-DNT for one year (Leonard et al. 1987). 2,6-DNT was mutagenic in the Ames Salmonella test strain TA100 (Spanggord et al. 1982), and induced framshift mutations in

strains TA1536, TA1537 and TA1538 (Tokiwa et al. 1981, Simmon et al. 1977). In rats, chronic exposure to pure 2,6-DNT and mixtures of 2,4- and 2,6-DNT in the diet have significantly increased the incidence of hepatocarcinoams (Ellis et al. 1979; Leonard et al. 1987).

EPA (1990) has classified 2,6-DNT in Group B2 --Probable Human Carcinogen for oral and inhalation routes. EPA (1990) has established an oral cancer potency factor of 0.68 $(mg/kg/day)^{-1}$ based on a 2-year study in which rats developed liver and mammary gland tumors when fed a mixture of 2,4- and 2,6- dinitrotoluene isomers (Ellis et al. 1979).

DI-*n*-OCTYL PHTHALATE

Di-*n*-octyl phthalate is not especially toxic. It is a severe eye and a mild skin irritant in rabbits (NIOSH 1985, NTP/IRLG 1982, EPA 1980). Subchronic dietary exposure to rodents has resulted in elevated kidney and liver weights, in addition to increased SGOT and SGPT (Piekacz 1971). Fetotoxicity and developmental abnormalities were observed in the offspring of rats administered 5 g/kg intraperitoneal injections on days 6 to 15 of gestation (NTP/IRLG 1982, EPA 1980).

EPA (1990) has derived an oral RfD of $2x10^{-2}$ mg/kg/day for di-n-octyl phthalate based on elevated kidney and liver weights and increased SGOT and SGPT in rats exposed to dietary concentrations of 175 mg/kg/day for 7-12 months (Piekacz 1971). A saftey factor of 1,000 was used to calculate the RfD.

ETHYLBENZENE

Ethylbenzene is absorbed via inhalation and distributed throughout the body in rats; the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al. 1980). In humans, short-term inhalation exposure to 435 mg/m^3 ethylbenzene for 8 hours can result in sleepiness, fatigue, headache, and mild eye and respiratory irritation (Bardodej and Bardodejova 1970); eye irritation has also been observed in experimental animals exposed to ethylbenzene (EPA 1987). Increased weights and cloudy swelling were observed in the liver and kidney of rats exposed to ethylbenzene by gavage at a dose of 408 mg/kg/day for 182 days (Wolf et al. 1956). A single oral dose of ethylbenzene administered to male and female Wistar-derived rats was reported to have an LD_{50} of 3,500 mg/kg body weight, with systemic effects occurring primarily in the liver and kidney (Wolf et al. 1956). Maternal toxicity was observed in rats exposed by inhalation to 4,348 mg/m^3

ethylbenzene for 6-7 hours/day during the first 19 days of gestation (Hardin et al. 1981). Ethylbenzene has also elicited developmental effects in experimental animals following inhalation exposure (Andrew et al. 1981, Hardin et al. 1981).

EPA (1991) derived an oral reference dose of 0.1 mg/kg/day for ethylbenzene based on the chronic study by Wolf et al. (1956) in which no liver or kidney effects were observed in rats exposed to 136 mg/kg/day. An uncertainty factor of 1,000 was applied to the no-observed-effect-level to derive the reference dose. EPA (1991) has also established an inhalation reference concentration (RfC) on 1 mg/m^3 for ethylbenzene based on developmental toxicity (Andrew et al. 1981, Hardin et al. 1981). This concentration is equivalent to a dose of $2.85x10^{-1}$ mg/kg/day, assuming a 70 kg individual inhales 20 m^3/day. An uncertainty factor of 300 was used to calcutate the RfC. EPA (1990) derived a subchronic oral RfD of 1 mg/kg/day using an uncertainty factor of 100 based on the same study and same effects of concern.

BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate, also known as di-ethylhexyl phthalate (DEHP), is readily absorbed following oral or inhalation exposure (EPA 1980). Chronic exposure to relatively high concentrations of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in laboratory animals (NTP 1982, EPA 1980, Carpenter et al. 1953). Reduced fetal weight and increased number of resorptions have been observed in rats exposed orally to DEHP (EPA 1980). DEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP 1982).

DEHP has been classified in Group B2--Probable Human Carcinogen (EPA 1986, 1991). EPA (1991) calculated an oral cancer potency factor for DEHP of $1.4x10^{-2}$ $(mg/kg/day)^{-1}$ based on data from the NTP (1982) study in which liver tumors were noted in mice. EPA has recommended an oral reference dose (RfD) for DEHP of $2x10^{-2}$ mg/kg/day for both chronic (EPA 1991) and subchronic (EPA 1991) exposures based on a study by Carpenter et al. (1953) in which increased liver weight was observed in female guinea pigs exposed to 19 mg/kg bw/day in the diet for 1 year (EPA 1991); an uncertainty factor of 1,000 was used to develop both RfDs.

HMX --

HMX, or octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine, is poorly absorbed when administered orally and intravenously to rats and mice due to its low aqueous solubility (EPA 1988). No data were found in the available literature regarding pulmonary or dermal absorption. Although no adverse effects were reported in workers who had been potentially exposed to HMX at a munitions plant, acute oral doses of HMX administered to rats and mice have resulted in histologic liver changes, and CNS effects including ataxia and hyperkinesia. Animals receiving higher doses experienced convulsions (EPA 1988). Subchronic oral administration of HDX to rats caused transient weight loss and blood changes (e.g. reduced hemoglobin, hematocrit, and red blood cell counts) in all treated animals (DOD, 1985). At higher HDX levels, males exhibited liver necrosis and enlarged centrilobular cells while tubular kidney changes such as focal atrophy and dilation were seen in treated female rats (DOD 1985). These results suggest a sex difference in target organs of rats to HMX (DOD 1985). Microbial genetic toxicology assays suggest that HMX is not mutagenic, although only low concentrations of HMX were used in tests due to limited solubility (DOD 1977).

EPA (1991) has reported an oral reference dose (RfD) of $5x10^{-2}$ mg/kg/day based on a subchronic rat feeding study where administration of 150 mg/kg/day led to hepatic lesions (DOD 1985). The RfD was determined by applying an uncertainty factor of 1,000 to the no observed adverse effect level (NOAEL) of 50 mg/kg/day for males.

IRON

Gastrointestinal absorption of iron in humans ranges from 1% to 25% (EPA 1984). Absorption of iron following inhalation exposure has not been thoroughly studied. Iron is an essential element and is therefore nontoxic at doses necessary for maintaining normal health and nutrition (EPA 1984). However, overexposure to iron can cause adverse health effects. Gastrointestinal irritation is the primary health effect observed in humans following acute oral overexposure to iron. In humans, chronic oral overexposure to iron has been associated with gastrointestinal bleeding, metabolic acidosis, hepatic toxicity, hemosiderosis, and hemochromatosis (EPA 1984). Human fatalities have occurred following ingestion of iron at doses of 100 mg/kg/day (Venugopal and Luckey 1978). Chronic inhalation overexposure of humans to iron-containing dusts and fumes produces respiratory irritation and various pulmonary lesions (EPA 1984). There is limited evidence from studies with experimental animals that certain soluble iron salts are teratogenic. Certain iron compounds are also

reported to be genotoxic (EPA 1984). Iron oxide enhances the carcinogenic action of various organic carcinogens (benzo[a]pyrene for example) and may act as a tumor promoter. Local sarcomas have been induced by subcutaneous injection of iron-dextran (EPA 1984).

The National Research Council of the National Academy of Sciences (NRC 1980) has suggested the recommended dietary allowances (RDAs) for iron of between 10 and 60 mg. Therefore, the maximum recommended daily intake of iron can be used as a conservative allowable intake for chronic exposure. No health based criteria have been derived by EPA.

LEAD

Absorption of lead from the gastrointestinal tract of adult humans is estimated at 8%-45%. In children, absorption from non-paint sources ranges from 30% to 50% (Hammond and Beliles 1980, EPA 1986). There are other interpretations of the data (Duggan 1983) that suggest this may be as high as 70%. For adult humans, the overall absorption rate is 30%-50%, however essentially all of the particulate airborne lead deposited in the lower respiratory tract is absorbed. Lead is stored in the body in the kidney, liver, and bone (EPA 1984). The major adverse effects in humans caused by lead include alterations in the hematopoietic and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Blood concentration levels of over 80 μg/dl in children and over 100 μg/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possible death. The Centers for Disease Control (CDC 1985) have used the value of 25 μg/dl as an acceptable level of blood lead. Recent information (EPA 1988), however, indicates that physiological and/or biochemical effects can occur even at lower levels. These include enzyme inhibition (16 μg/dl), elevated erythrocyte protoporphyrin (15 μg/dl), interference with Vitamin D metabolism, cognitive dysfunction in infants (10 to 15 μg/dl), electrophysiological dysfunction (6 μg/dl), and reduced childhood growth (4 μg/dl). Decreased fertility, fetotoxic effects, and skeletal malformations have been observed in experimental animals exposed to lead (EPA 1984). Chronic oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated in experimental animals with increased renal tumors. Doses of lead that induced kidney tumors were high and were beyond the lethal dose in humans (EPA 1985).

EPA classified certain lead salts in Group B2 (Probable Human Carcinogen), although no cancer potency factor has been established (EPA 1991). This category applies to those agents for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in

humans. EPA (1988) has proposed a maximum contaminant level goal (MCLG) of zero for lead. EPA (1991) has considered it inappropriate to develop a reference dose (RfD) for inorganic lead and lead compounds, since many of the health effects associated with lead intake (particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development) occur essentially without a threshold. The new proposed MCLG is based on subtle effects of lead at low blood levels, the overall Agency goal of reducing lead exposures, and the probable carcinogenicity of lead at very high doses. Underlying this proposal was the assumption that blood lead levels in the range of 10-15 μg/dl are associated with serious effects. Additionally EPA noted that existing body burdens of lead were already in the range where adverse effects could result.

An alternative approach is also undergoing review by EPA to evaluate potential subchronic lead exposures to young children. This approach is based on a linear pharmacokinetic model used by EPA's Office of Air Quality Planning and Standards (OAQPS) for lead air quality standard setting (EPA 1989). The model, based on work by Harley and Kneip (1985), takes into account the uptake, retention and excretion of lead. It is referred to as the "Integrated Uptake/Biokinetic Model", and it estimates blood lead levels.

MAGNESIUM

Magnesium is an essential nutrient which acts as a cofactor for many enzymes and plays an important role in neurological transmission and muscular excitability. Absorption of magnesium following oral exposures is dose-dependent in both humans and animals. At physiological doses of 3 to 10 milliequivalent (mEq), less than 10% appeared in the urine in 72 hours, while 59-88% was excreted in the feces in 120 hours (Stokinger 1981). An average adult in the United States ingests between 20 and 40 mEq of magnesium per day, one third of which is absorbed primarily in the small intestine. Normal magnesium plasma levels in humans range between 1.5 to 2.2 mEq per liter (Goodman and Gilman 1985). In humans, acute toxicity from inhalation of magnesium oxide fume results in metal fume fever (Sax 1984) and associated leukocytosis. Excessive magnesium can cause muscle weakness, hypotension, sedation, confusion, respiratory paralysis, coma and death in humans. Human exposure to particles of magnesium in subcutaneous tissue produce lesions that resist healing (Goyer 1986) and will cause skin and eye burns since it reacts with water to form caustic magnesium hydroxide (National Fire Protection Assoc. 1978). The oral lethal dose (LD_{50}) for dogs is 230 mg/kg. Dermal applications of magnesium powder to abraded surfaces of animals results in an inflammatory reaction (Stokinger 1981). Limited data are available on magnesium's carcinogenic potential. One

study found intratracheal administration to hamsters produced olfactory tumors, and tumors of the lungs, thorax and respiratory system (RTECS 1987). No health-based criteria have been established by EPA.

MANGANESE

Manganese is absorbed at low levels following oral or inhalation exposure (EPA 1984a). The effects following acute exposure to manganese are unknown. Chronic oral and inhalation exposure of humans to high levels of manganese causes pneumonitis in exposed workers and has been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking (Kawamura et al. 1941). Altered hematologic parameters (hemoglobin concentrations, erythrocyte counts) have also been observed in individuals exposed chronically. Chronic oral exposure of rats to manganese chloride can result in central nervous system dysfunction (Leung et al. 1981, Lai et al. 1982). Manganese has not been reported to be teratogenic; however, this metal has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (EPA 1984a). Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinomal cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (EPA 1984a,b).

EPA (1991) established an oral reference dose (RfD) of 1.0×10^{-1} mg/kg/day for manganese based on a no-observed-adverse-effects level (NOAEL) of 0.14 mg/kg/day in humans chronically exposed to manganese in food (WHO 1973, Schroeder et al. 1966, NRC 1989). An uncertainty factor of 1 was used to derive the reference dose. EPA (1990) calculated an inhalation reference dose based upon an occupational study conducted by Saric et al. (1977) examining central nervous effects of manganese. Using a NOAEL of 2.1 mg/day and an uncertainty factor of 100, an inhalation RfD of 3.0×10^{-4} mg/kg/day was derived. Both the inhalation and oral values are based upon central nervous system effects (EPA 1991, 1990).

MERCURY

In humans, inorganic mercury is absorbed following inhalation and oral exposure, however only 7% to 15% of administered inorganic mercury is absorbed following oral exposure (EPA 1984, Rahola et al.

1971, Task Group on Metal Accumulation 1973). In humans, organic mercury is almost completely absorbed from the gastrointestinal tract and is assumed to be well absorbed via inhalation in humans (EPA 1984). A primary target organ for inorganic compounds is the kidney. Acute and chronic exposures of humans to inorganic mercury compounds have been associated with anuria, polyuria, proteinuria, and renal lesions (Hammond and Beliles 1980). Chronic occupational exposure of workers to elemental mercury vapors (0.1 to 0.2 mg/m^3) has been associated with mental disturbances, tremors, and gingivitis (EPA 1984). Animals exposed to inorganic mercury for 12 weeks have exhibited proteinuria, nephrotic syndrome and renal disease (Druet et al. 1978). Rats chronically administered inorganic mercury (as mercuric acetate) in their diet have exhibited decreased body weights and significantly increased kidney weights (Fitzhugh et al. 1950). The central nervous system is a major target for organic mercury compounds. Adverse effects in humans, resulting from subchronic and chronic oral exposures to organic mercury compounds, have included destruction of cortical cerebral neurons, damage to Purkinje cells, and lesions of the cerebellum. Clinical symptoms following exposure to organic mercury compounds have included paresthesia, loss of sensation in extremities, ataxia, and hearing and visual impairment (WHO 1976). Embryotoxic and teratogenic effects, including malformations of the skeletal and genitourinary systems, have been observed in animals exposed orally to organic mercury (EPA 1984). Both organic and inorganic compounds are reported to be genotoxic in eukaryotic systems (Leonard et al. 1984).

EPA (1990) has reported an oral reference dose for both chronic and subchronic exposures of $3x10^{-4}$ mg/kg/day for inorganic mercury based on several oral and parenteral studies conducted in the Browwn Norway rat studies which observed kidney effects (Fitzhugh et al. 1950, Druet et al. 1978, Bernaudin et al. 1981). An uncertainty factor of 1,000 was used to derive the RfDs. EPA (1990) has also derived an inhalation RfC for inorganic mercury of $3x10^{-4}$ mg/m^3 for both chronic and subchronic exposures based on several human occupational studies in which neurotoxicity was observed (Fawer et al. 1987, Piikivi and Tolonen 1989, Piikivi and Hanninen 1989, Piikivi 1989). This is equivalent to a dose of $8.5x10^{-5}$ mg/kg/day assuming a 70 kilogram individual inhales 20 m^3/day. An uncertainty factor of 30 was used to derive both inhalation RfCs.

NICKEL

Nickel compounds can be absorbed following inhalation, ingestion, or dermal exposure. The amount absorbed depends on the dose administered and the chemical and physical form of the particular

nickel compound (EPA 1986). Dermal exposure of humans to nickel produces allergic contact dermatitis (EPA 1986). Adverse effects associated with acute exposure in animals have included depressed weight gain, altered hematological parameters, and increased iron deposition in blood, heart, liver, and testes (EPA 1987). Chronic or subchronic exposure of experimental animals to nickel has been associated with reduced weight gain, degenerative lesions of the male reproductive tract, asthma, nasal septal perforations, rhinitis, sinusitis, hyperglycemia, decreased prolactin levels, decreased iodine uptake, and vasoconstriction of the coronary vessels (EPA 1986). Teratogenic and fetotoxic effects have been observed in the offspring of exposed animals (EPA 1986). Inhalation exposure of experimental animals to nickel carbonyl or nickel subsulfide induces pulmonary tumors (EPA 1986). Several nickel salts cause localized tumors when administered by subcutaneous injection or implantation. Epidemiological evidence indicates that inhalation of nickel refinery dust and nickel subsulfide is associated with cancers of the nasal cavity, lung, larynx, kidney, and prostate (EPA 1986).

Nickel refinery dust and nickel subsulfide are both categorized as Group A carcinogens (Human Carcinogen) (EPA 1991). These classifications are based on an increased incidence of lung and nasal tumors observed in workers occupationally exposed to nickel refinery dust (EPA 1986). These materials have inhalation cancer potency factors of 0.84 $(mg/kg/day)^{-1}$ and 1.7 $(mg/kg/day)^{-1}$, respectively (EPA 1991). Nickel carbonyl is categorized in Group B2 (Probable Human Carcinogen); however, a potency factor has not been derived for nickel carbonyl (EPA 1991). EPA derived an oral reference dose (RfD) for nickel of $2x10^{-2}$ mg/kg/day for both chronic (EPA 1991) and subchronic (EPA 1990) exposures based on a study by Ambrose et al. (1976) in which rats administered 5 mg/kg/day (NOAEL) nickel in the diet for 2 years did not experience decreased weight gain which was observed in animals administered 50 mg/kg/day (LOAEL). A safety factor of 300 was used to calculate the oral RfDs.

NITROBENZENE

Nitrobenzene is absorbed by all possible routes, but absorption primarily occurs through the respiratory tract and skin (EPA 1980); approximately 80% of inhaled nitrobenzene is absorbed (EPA 1980). In humans, long-term occupational exposure to nitrobenzene can result in cyanosis, methemoglobinemia, jaundice, anemia, sulfhemoglobinemia, and dark urine (EPA 1980). Short-term exposure to high levels of nitrobenzene can result in cyanosis, and if severe, the individual can go into a coma (Piotrowski 1967). Hematologic, adrenal, renal, and hepatic lesions have been reported in rats

and mice exposed to nitrobenzene in air for 90 days (CIIT 1984). There is also limited evidence that exposure to nitrobenzene can result in changes in the tissues of the chorion and placenta in pregnant women (Dorigan and Hushon 1976); menstrual disturbances after chronic nitrobenzene exposure have also been reported (EPA 1980).

EPA (1990) developed an inhalation RfD of $2x10^{-3}$ mg/m^3 for nitrobenzene based on a study in which hematological, adrenal, renal, and hepatic lesions were observed in mice following inhalation exposure to nitrobenzene (CIIT 1984); an uncertainty factor of 3,000 was used in the derivation. This concentration is equivalent to a dose of $5.7x10^{-4}$ mg/kg/day assuming a 70 kilogram individual inhales 20 m^3/day. EPA (1991) developed an oral RfD for nitrobenzene of $5x10^{-4}$ mg/kg/day based on the same CIIT (1984) study, using route-to-route extrapolation, the same endpoints of toxicity and an uncertainty factor of 10,000.

POTASSIUM

Absorption of dietary potassium from the gastrointestinal tract is nearly complete (Goodman and Gilman 1985). Potassium is a reactive compound that is strongly caustic and corrosive upon contact with tissues (Wands 1981). Potassium is an essential element and more concern is generally associated with potassium deficiency (particularly in the elderly) than with toxicity. It functions in the maintenance of electrolyte balance, in the transmission of nerve impulses to muscle fibers, in the control of normal muscle contractility and cardiac rhythm, and it acts as an insulin antagonist in intermediary carbohydrate metabolism (NRC 1980). Ingestion of excess potassium results in hyperkalemia which alters the electrical activity of the heart. At potassium levels of 8 to 9 mEq per liter, there is profound depression in impulse generation and conduction in all cardiac tissues (Goodman and Gilman 1985). The National Research Council of the National Academy of Sciences (NRC 1980) has determined that the estimated adequate and safe intake level for potassium is between 1,875 and 5,600 mg/day for adults. NRC (1980) also noted that "it is not possible to induce hyperkalemia or potassium toxicity by dietary means in people with normal circulatory and renal function." However, acute poisoning with potassium chloride tablets has been observed in children at potassium levels as low as 2,000 mg/day (NRC 1980). No health-based criteria have been established for potassium by EPA.

RDX

RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine; cyclonite) is completely absorbed following oral exposure (EPA 1988). No data are available regarding dermal absorption. Workers exposed to RDX via inhalation and gastrointestinal routes suffered CNS effects, including headaches, nausea, vomiting, amnesia, clonic/tonic convulsions, and unconsciousness (Gosselin 1984; Kaplan 1965). These symptoms paralleled those previously reported in animal studies (Sunderman et al 1944; Von Oettingen et al 1949). However, a cross-sectional epidemiological study in a munitions plant did not identify any abnormalities in employees attributable to RDX exposure (Hathaway 1977). In acute toxicity studies, dogs exposed intravenously to RDX experienced decreased blood pressure and erratic electroencephalographic patterns at low doses, central nervous system hyperactivity and nonlethal convulsions at higher doses, and convulsions and death at the highest dose levels (EPA 1988). In subchronic feeding studies, mice experienced increased liver weights. Anemia was seen in male mice and rats, and female rats experienced increased liver weights (EPA 1988). Chronic oral exposure to RDX in rats and mice produced CNS effects, increased mortality, weight loss, anemia, hepatoxicity, renal toxicity, testicular degeneration, and inflammation of the prostate (Levine et al. 1983; EPA 1988). Decreased fertility, developmental effects, and embryotoxicity were observed in rats that were fed RDX. In rabbits, RDX caused maternal toxicity, and there was suggestive evidence for teratogenic effects (EPA 1988). No conclusive evidence of carcinogenicity has been shown for RDX. RDX was not found to be carcinogenic in Fisher 344 rats (Levine et al. 1983) or Sprague-Dawley rats (Hart 1977) exposed to RDX in the diet for 2 years. However, Lish et al. (1984) reported a statistically significant increase in the combined incidence of hepatocellular carcinomas and adenomas in female B6C3F1 mice fed RDX in the diet for two years.

EPA (1991) has classified RDX in Group C -- Possible Human Carcinogen -- and has developed an oral cancer potency factor of 0.11 $(mg/kg/day)^{-1}$. The potency factor is based on the increased incidence of combined hepatocellular carcinomas and adenomas in female mice receiving RDX in the diet for two years (Lish et al. 1984). EPA (1991) has derived a reference dose (RfD) of $3x10^{-3}$ mg/kg/day based on a chronic study in which rats receiving RDX in the diet for 24 months at varying dosages experienced inflammation of the prostate (Levine et al. 1983). A lowest observed adverse effect level (LOAEL) of 1.5 mg/kg/day was identified. An uncertainty factor of 100 was used to derive the RfD.

SILVER

Silver in various forms is absorbed to a limited extent following oral and inhalation exposures (EPA 1985). The acute toxic effects in humans following oral exposure to silver include corrosive damage to the GI tract leading to shock, convulsions, and death. In animals, acute exposure has been shown to affect the central nervous system and to cause respiratory paralysis (Hill and Pillsbury 1939). The primary effect of silver in humans following chronic exposures is argyria, a permanent bluish-metallic discoloration of the skin and mucous membranes, which can be either localized or generalized. Silver also accumulates in the blood vessels and connective tissue (EPA 1985).

EPA (1991) derived an oral reference dose (RfD) for silver of 3.0×10^{-3} mg/kg/day for both chronic (1991) and subchronic (EPA 1990) exposures based on the human case reports of Gaul and Staud (1935), Blumberg and Carey (1934), and East et al. (1980). In these studies, argyria was observed at an average dose of silver of 0.0052 mg/kg/day, to which an uncertainty factor of 2 was applied.

SODIUM

Sodium is rapidly and almost completely absorbed from the gastrointestinal tract (NAS 1977). In humans, adverse effects of sodium occur as a result of ingestion of excess amounts of this element. Acute effects appear to occur only in neonates and young infants. Several studies suggest that permanent brain damage and sudden, unexpected deaths of infants between the ages of 2 weeks and 2 years may be due to hypernatremia (NAS 1977, NIOSH 1987, NRC 1980). Clinical and epidemiological studies suggest that prolonged ingestion of excess sodium may contribute to the development of hypertension in genetically susceptible people (NAS 1977, Wands 1981). In humans, sodium vapors and fumes are strongly alkaline and are extremely irritating and corrosive to the respiratory tract, eyes and skin (Wands 1981). Studies with experimental animals support the contention that excess sodium ingestion is related to the development of hypertension (NAS 1977). It is estimated that at least 40 percent of the population would benefit if consumption of sodium were limited to 2,000 mg/kg or less (EPA 1985, NAS 1977, NRC 1986). Sodium is reported to produce teratogenic and reproductive effects in experimental animals exposed to high doses by various routes. For example, mice exposed subcutaneously to over 2,000 mg/kg of sodium chloride on day 10 or 11 of gestation had an increased incidence of dead or resorbed young. Live young in this study had decreased body weights and an increased incidence of appendicular malformations, such as clubfoot

and deviation of the digits (Nishimuri and Miyamoto 1969). No health-based criteria have been developed by EPA.

1,1,2,2-TETRACHLOROETHANE

In humans, absorption of a single inhalation dose of 1,1,2,2,-tetrachloroethane vapor was reported to be 97%; absorption of this chemical from the gastrointestinal tract is inferred from studies in which an increased incidence of liver tumors was reported in mice exposed in the diet (EPA 1984). The effects associated with occupational exposure to 1,1,2,2-tetrachloroethane by inhalation or dermal routes are primarily neurological and include, tremors, headache, numbness, excessive perspiration, and anorexia (EPA 1984). In experimental animals, subchronic inhalation exposure to 1,1,2,2-tetrachloroethane is associated with liver effects, decreased hemoglobin content of red blood cells, decreased hematocrit, and fluctuations in white blood cell count (Schmidt et al. 1972, Navrotskiy et al. 1971, Horiuchi et al. 1962). 1,1,2,2-Tetrachloroethane is a liver carcinogen when administered orally to mice (NCI 1978).

EPA (1991) classified 1,1,2,2-tetrachloroethane in Group C--Possible Human Carcinogen based on increased incidence of hepatocellular carcinoma in mice. EPA (1991) developed an oral cancer potency factor of 0.2 $(mg/kg/day)^{-1}$ based on the study conducted by NCI (1978) in which a highly significant dose-related increase in the incidence of hepatocellular carcinomas was observed in both male and female mice. An inhalation cancer potency factor of 0.2 $(mg/kg/day)^{-1}$ was also calculated from these data (EPA 1991). EPA (1987) has also derived an interim oral reference dose (RfD) of $4.6x10^{-4}$ mg/kg/day for 1,1,2,2-tetrachloroethane based on an inhalation study by Schmidt et al. (1972) in which rats were exposed to 1,1,2,2-tetrachloroethane vapor for 5 hours/day for 265 days. In this study decreased body weight, increased white blood cell count and increased hepatic fat content were observed. Using a LOAEL of 0.456 mg/kg/day and applying a safety factor of 1,000 the interim RfD was derived.

TETRYL

Tetryl is absorbed through oral, inhalation, and dermal routes. It is a skin and mucous membrane irritant, with the most common reaction being skin sensitization and dermatitis. Industrial exposure to tetryl has caused severe upper respiratory tract irritation with coughing and epistaxis, edema, headache, irritability, malaise, lassitude, sleeplessness, and conjunctivitis (Witkowski et al. 1942).

Dermal exposure may stain the skin and hair yellow (Hamilton and Hardy 1974). Additionally, heavy airborne exposure to tetryl may cause liver damage (Hardy and Maloof 1950; Schwartz 1942). Acute exposure to tetryl in rabbits (via gavage) and dogs (subcutaneously) led to severe acute inflammation at the injection site, varying degrees of edema and hemorrhaging, degeneration of muscle tissue, and toxic degeneration of the kidneys. Dog livers showed varying degrees of necrosis in the centers of the lobules and severe fatty degeneration of the liver cells. Rabbit livers showed almost no changes (Wells 1920). Tetryl appears to be a potent, direct-acting mutagen in three microbial mutagenicity test systems (*Neurospora crassa, Salmonella typhimurium, and Saccharomyces cerevisiae* D_4) even at low concentrations (Whong et al. 1980). EPA has not derived health-based criteria for tetryl.

TOLUENE

Toluene is absorbed in humans following both inhalation and dermal exposure (EPA 1985). In humans, the primary acute effects of toluene vapor are central nervous system (CNS) depression and narcosis. These effects occur at concentrations of 200 ppm (754 mg/m^3) (von Oettingen et al. 1942a,b). In experimental animals, acute oral and inhalation exposures to toluene can result in central nervous system (CNS) depression and lesions of the lungs, liver, and kidneys (EPA 1987). The earliest observable sign of acute oral toxicity in animals is depression of the CNS, which becomes evident at approximately 2,000 mg/kg (Kimura et al. 1971). In humans, chronic exposure to toluene vapors at concentrations of approximately 200 and 800 ppm has been associated with CNS and peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (EPA 1987, Anderson et al. 1983). Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure (Hanninen et al. 1976, von Oettingen et al. 1942a). In rats, chronic exposure to toluene via inhalation results in CNS toxicity and a dose-related reduction in hematocrit values (CIIT 1980). The liver and kidney (NTP 1989a) are the target organs in rats following prolonged gavage administration (NTP 1989a). There is some evidence in mice that oral exposure to greater than 0.3 ml/kg toluene during gestation results in embryotoxicity (Nawrot and Staples 1979). Inhalation exposure of up to 1,000 mg/m^3 by pregnant rats during gestation has been associated with significant increases in skeletal retardation (Hudak and Ungvary 1978).

EPA (1991) has derived an oral risk reference dose (RfD) of 0.2 mg/kg/day for toluene based on a gavage study in which rats were given doses as high as 5,000 mg/kg 5 days/week for 13 weeks and changes in liver and kidney weights were observed (NTP 1989). No adverse effects were observed in any of the treated animals at 223 mg/kg/day (NOAEL). An uncertainty factor of 1,000 was used to

calculate the oral RfD. EPA (1990) derived an oral subchronic RfD of $4x10^{-1}$ based on a gavage study in which rats experienced CNS effects (Wolf et al. 1956). An uncertainty factor of 100 was used to calculate the RfD. EPA (1990) reported chronic and subchronic inhalation RfDs for toluene of 2 mg/m^3 which is equivalent to $5.7x10^{-1}$ mg/kg/day, assuming a 70 kilogram individual inhales 20 m^3/day, based on the development of adverse CNS effects in humans (Anderson et al. 1983). An uncertainty factor of 100 was used for both exposures.

1,1,2-TRICHLOROETHANE

1,1,2-Trichloroethane (1,1,2-TCA) is rapidly absorbed from oral, inhalation and dermal exposures (Torkelson and Rowe 1981, Arena 1979). In humans, acute oral and inhalation exposures to 1,1,2-TCA result in central nervous system (CNS) depression, equilibrium disturbances, vertigo, headaches, lassitude, hypotension, anesthesia and coma (Arena 1979). Acute oral and inhalation administration to animals produces liver and kidney damage, irritation to the eyes and nose, CNS depression, and death due to respiratory arrest (ACGIH 1986, Torkelson and Rowe 1981). In dogs the hepatotoxic effects include hepatocyte vacuolation, enzyme induction, fatty degeneration and necrosis (NRC 1977, Torkelson and Rowe 1981). The hepatoxicity and nephrotoxicity of 1,1,2-TCA has been found to be potentiated by pretreatment with certain halogenated organic compounds and solvents. Subchronic oral administration to mice produced alterations in clinical serum levels indicative of adverse liver effects (White et al. 1985, Sanders et al. 1985). Dermal exposures result in irritation and injury to the skin from defatation (Torkelson and Rowe 1981). Evidence suggests that 1,1,2-TCA is embryo toxic to chicken eggs (Elovaara 1979). 1,1,2-TCA was found to be weakly mutagenic in S. Cerevisiae (Torkelson and Rowe 1981). Oral administration of 1,1,2-TCA has been associated with the induction of hepatocellular carcinomas and pheochromocytomas in mice but not in rats (NCI 1978, Weisburger 1977)

EPA has classified 1,1,2-TCA in group C (Possible Human Carcinogen). This category applies to agents for which there is limited evidence of carcinogenicty in animals. EPA (1991) has derived a cancer potency factor of $5.7x10^{-2}$ $(mg/kg/day)^{-1}$ for both oral and inhalation exposures based on an increased incidence of liver tumors in mice (NCI 1978). EPA (1991) has also established an oral reference dose (RfD) of $4.0x10^{-3}$ mg/kg/day for 1,1,2-TCA based upon clinical chemistry alterations in mice given 3.9 mg/kg/day in drinking water (White et al 1985, Sanders et al 1985). An uncertainty factor of 1,000 was used to calculate the RfD. EPA (1990) has derived a subchronic oral RfD of

$4.0x10^{-2}$ mg/kg/day, using an uncertainty factor of 100, and based on the same study and effect of concern.

TRICHLOROETHYLENE

Absorption of trichloroethylene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (EPA 1985). TCE is a central nervous system depressant following acute and chronic exposures. In humans, single oral doses of 15 to 25 ml (21 to 35 grams) of TCE have resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephens 1945). High-level exposure can result in death due to respiratory and cardiac failure (EPA 1985). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (EPA 1985). Nephrotoxicity has been observed in animals following acute exposure to TCE vapors (ACGIH 1986, Torkelson and Rowe 1981). Subacute inhalation exposures of mice have resulted in transient increased liver weights (Kjellstrand et al. 1983). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported following exposure to TCE in dilute, aqueous solutions (EPA 1985). Trichloroethylene has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI 1976) and renal tubular-cell neoplasms in rats exposed by gavage (NTP 1983), and pulmonary adenocarcinomas in mice following inhalation exposure (Fukuda et al. 1983, Maltoni et al. 1986). Trichloroethylene was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim et al. 1977).

EPA (1990) classified trichloroethylene in Group B2--Probable Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. An oral cancer potency factor of $1.1x10^{-2}$ $(mg/kg/day)^{-1}$ has been derived by EPA (1990) based on two gavage studies conducted in mice in which an increased incidence of liver tumors were observed (Maltoni et al. 1986, Fukuda et al. 1983). An inhalation cancer potency factor of $1.7x10^{-2}$ $(mg/kg/day)^{-1}$ has been derived for trichloroethylene (EPA 1990) based on an increased incidence of lung tumors in mice (NCI 1976). EPA (1987) developed an oral reference dose (RfD) of $7.35x10^{-3}$ mg/kg/day based on a subchronic inhalation study in rats in which elevated liver weights were observed following exposure to 55 ppm, 5 days/week for 14 weeks (Kimmerle and Eben 1973). A safety factor of 1,000 was used to calculate the RfD. However, this RfD is currently under review by EPA.

TRICHLOROFLUOROMETHANE

Trichlorofluoromethane is rapidly absorbed in humans and animals following oral and inhalation exposures (ACGIH 1986, Mergner et al. 1975, Adir et al. 1975). Oral administration of 2.5 ml/kg in rats resulted in neither death or liver toxicity (Slater 1965) and application to the eyes or skin caused only mild reversible irritation (Scholz 1962, Quevauviller et al. 1964, Hood 1964). The LC_{50} of trichlorofluoromethane in rats after a 4 hour exposure has been determined to be 26,200 ppm (ACGIH 1986). Metabolic changes (e.g., increased blood glucose and lactic acid, decreased oxygen uptake) were reported in rats and rabbits exposed to 50,000 ppm trichlorofluoromethane for one hour daily for 15 days (Paulet 1975). Dogs exposed to the same concentration for 20 minutes showed increases in blood glucose and lactic acid. A subchronic study using dogs exposed to trichlorofluoromethane in the air for 90 days showed elevated BUN levels and lung lesions (Jenkins et al. 1970). Evidence of cardiotoxicity (cardiac arrhythmias) in dogs exposed to 5000 ppm trichloromethane and intravenous epinephrine was provided by Reinhardt et al. (1971). Subsequent experiments have shown the effects to be brief with the animals recovering shortly after the end of exposure (Clark et al. 1972). Other animals exposed to trichlorofluoromethane concentrations between 5,000 to 15,00 ppm have also been shown to exhibit cardiovascular and circulatory abnormalities (Flowers et al. 1975, Taylor and Drew 1975, Taylor 1975, Aviado 1975). The available evidence suggests that trichlorofluoromethane is not carcinogenic (NCI 1978). This study did show an association between dose and increased incidence of mortality in rats.

EPA (1990) has reported chronic and subchronic inhalation RfDs for trichlorofluoromethane of 0.2 mg/kg/day and 2 mg/kg/day, respectively, based on studies investigating elevated BUN levels and lung lesions in dogs (Jenkins et al. 1970); uncertainty factors of 10,000 and 1,000 were used to develop the RfDs for chronic and subchronic exposures, respectively. A chronic (EPA 1991) and subchronic (EPA 1990) oral reference doses of 0.3 mg/kg/day and 0.7 mg/kg/day have been reported, respectively, for trichlorofluoromethane based on a chronic rat study in which increased mortality was observed (NCI 1978). An uncertainty factor of 1,000 was used to derive both the chronic and subchronic RfDs.

1,3,5-TRINITROBENZENE

Little information is available for 1,3,5-trinitrobenzene (TNB). Therefore, toxicity information is based on the structurally similar 1,3-dinitrobenzene (RTECS 1983). 1,3-dinitrobenzene is absorbed orally. In

subchronic drinking water studies, rats experienced decreased hemoglobin concentrations, splenic weight gain and enlargement with hemosiderin deposits, decreased body weight gain in females, and testicular atrophy in males in the two highest dose levels (Cody et al. 1981). 1,3,5-TNB has been shown to be mutagenic in DNA repair assays (McGregor et al. 1980).

EPA (1991) has derived an oral reference dose (RfD) for 1,3,5-TNB of $5x10^{-5}$ mg/kg/day based on a subchronic study whereby rats experienced increased spleen weights when given 1,3-dinitrobenzene in drinking water (Cody et al. 1981). The RfD for 1,3,5-TNB was derived by analogy from 1,3-dinitrobenzene. A NOAEL of 0.4 mg/kg/day for 1,3-DNB (equivalent to 0.51 mg/kg/day 1,3,5-TNB) was identified from this study. An uncertainty factor of 10,000 was used to calculate the RfD for 1,3,5-TNB.

2,4,6-TRINITROTOLUENE (TNT)

2,4,6-Trinitrotoluene (TNT) is absorbed by humans and laboratory animals following oral, inhalation and dermal exposure (EPA 1989). Once absorbed, TNT is distributed throughout the body and is metabolized extensively (EPA 1980). The hematopoietic system and the liver are the principle target organs of TNT toxicity in humans and experimental animals (Levine et al. 1983; NRC 1982). Increased incidences of cataracts (Harkonen et al. 1983), neurological manifestations, and nephrotoxicity have been observed in humans occupationally exposed to TNT (Kaganov et al. 1970, as cited in Zakhari and Villaume 1978). Acute exposure in rats is associated with respiratory paralysis, cyanosis, ataxia, and sometimes death (Lee et al. 1975). Testicular atrophy and degeneration have been observed in rats subchronically exposed to TNT in the diet (Dilley et al 1982). TNT has been found to cause urinary bladder papillomas and carcinomas in female rats (U.S. DOD 1984). Mutagenic activity was observed in strains TA98, TA1537, TA1538, and TA100 of *Salmonella typhimurium* with and without metabolic activation (Spanggord et al. 1982).

EPA (1991) has assigned TNT a weight-of-evidence classification of C -- Possible Human Carcinogen -- based on urinary bladder papillomas and carcinomas at 50 mg/kg/day in female Fischer 344 rats (U.S. DOD 1984). An oral cancer potency factor of $3.0x10^{-2}$ $(mg/kg/day)^{-1}$ has been assigned to TNT. EPA (1991) has developed a reference dose (RfD) for TNT based on a study in beagle dogs in which an increased incidence of liver effects was observed (Levine et al. 1983). Animals received TNT in gelatin at daily oral doses of 0, 0.5, 2.0, 8.0 or 32 mg/kg/bw for 26 weeks. Gross and histologic examination revealed toxic liver effects and/or lesions in all dose groups with lesions in the low dose

group (0.5 mg/kg/day) described as trace to mild. No lesions were observed in the control animals. The 0.5 mg/kg/day dose is the lowest-observed-adverse-effect level (LOAEL) for this study. An RfD of $5.00x10^{-4}$ mg/kg/day was derived based on this LOAEL and an uncertainty factor of 1,000.

VANADIUM

Pentavalent vanadium compounds are generally considered to be more toxic than other valence states. Many incidents of short-term and long-term occupational exposures to vanadium, mainly vanadium pentoxide dust, have been reported. Inhalation causes respiratory tract irritation, coughing, wheezing, labored breathing, bronchitis, chest pains, eye and skin irritation and discoloration of the tongue (NIOSH 1977, NAS 1974). Effects seen in experimental animals following chronic inhalation exposure include fatty degeneration of the liver and kidneys, hemorrhage, and bone marrow changes (Browning 1969).

EPA (1990) has derived an oral reference dose (RfD) of $7x10^{-3}$ mg/kg/day based on a chronic study in which rats received vanadium in their drinking water (Schroeder et al. 1970). A no-observed-adverse-effect level (NOAEL) of 0.77 mg/kg/day and an uncertainty factor of 100 were used to develop the RfD. EPA (1991) has established an oral RfD for vanadium pentoxide of $9x10^{-3}$ mg/kg/day. This value is based on a chronic rat study in which a NOAEL of 0.89 mg vanadium pentoxide/kg/day was noted. The only reported effect was a decrease in the amount of cystine in the hair (Stokinger et al. 1953). An uncertainty factor of 100 was used to calculate the vanadium pentoxide RfD. EPA has not developed inhalation criteria for vanadium.

XYLENES

The three xylene isomers, compounds that have the same chemical constituents in a different configuration, have similar toxicological properties and are discussed together. Data from animals and humans suggest that approximately 60% of an inhaled dose is absorbed. Inference from metabolism and excretion studies suggests that absorption of orally administered xylene is nearly complete. Dermal absorption is reported to be minor following exposure to xylene vapor but may be significant following contact with the liquid (EPA 1985). In humans, acute inhalation exposures to relatively high concentrations of xylene adversely affect the central nervous system and lungs and can irritate mucous membranes (EPA 1987, Hake et al. 1981). Savolainen et al. (1980) observed that body balance and manual coordination were impaired in eight male students following inhalation exposure

to m-xylene. However, tolerance against the observed effects developed during one work week. In experimental rats, long-term inhalation exposure to o-xylene resulted in hepatomegaly (Tatrai et al. 1981). Oral exposure to 200 mg/kg xylene in the diet for up to 6 months was also associated with liver toxicity, specifically the development of intracellular vesicles (Bowers et al. 1982). Prolonged oral exposures in mice resulted in hyperactivity, a manifestation of CNS toxicity (NTP 1986). Xylene appears to be fetotoxic and may increase the incidence of visceral and skeletal malformations in offspring of exposed experimental animals (Mirkova et al. 1983). There is suggestive evidence that xylene is carcinogenic in experimental animals when exposed by oral gavage (Maltoni et al. 1985).

EPA (1991) calculated a chronic oral reference dose (RfD) for mixed xylenes of 2 mg/kg/day based on an NTP (1986) study in which male rats given a gavage dose of 179 mg/kg/day for 103 weeks did not exhibit hyperactivity, decreased body weight or significant increased mortality. The oral RfD was derived using the no-observed-adverse-effect level (NOAEL) of 179 mg/kg/day and an uncertainty factor of 100. EPA (1990) calculated a subchronic oral RfD of 4 mg/kg/day for mixed xylenes based on a 13 week gavage study where no adverse effects were seen in rats. An uncertainty factor of 100 was used to calculate the RfD. EPA (1990) reported a chronic and subchronic inhalation RfD for mixed xylenes of 0.3 mg/m^3 based on a study in which CNS effects, and nose and throat irritation were observed in humans exposed to 20 ppm (27 mg/m^3) for 5 days (Hake et al. 1981, Carpenter et al. 1975); an uncertainty factor of 100 was used to develop the RfD for both exposure levels. This concentration is equivalent to $8.6x10^{-2}$ mg/kg/day, assuming a 70 kilogram individual inhales 20 m^3/day.

ZINC

Zinc is absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (EPA 1984). Zinc is an essential trace element that is necessary for normal health and metabolism and therefore is nontoxic in trace quantities (Hammond and Beliles 1980). Exposure to zinc at concentrations that exceed recommended levels has, however, been associated with a variety of adverse effects. In humans, chronic and subchronic inhalation exposure to zinc has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea, and muscle pain (EPA 1984). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters (Pories et al. 1967, Prasad et al. 1975). Reduced body weights have been observed in studies in

which rats were administered zinc in the diet. There is no evidence that zinc is teratogenic or carcinogenic (EPA 1984).

EPA (1990) has derived an oral reference dose (RfD) of $2x10^{-1}$ mg/kg/day for both chronic and subchronic exposures based on studies in which anemia and reduced blood copper were observed in humans exposed to oral zinc doses of 2.14 mg/kg/day (Pories et al. 1967, Prasad et al. 1975). A safety factor of 10 was used to develop the RfD.

REFERENCES

Acetone

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Acetone. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-016

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Ninety-Day Gavage Study in Albino Rats Using Acetone. Office of Solid Waste, Washington, DC. (As cited in EPA 1989)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

GOLDBERG, M.E., JOHNSON, H.E., POZZANI, N.C., and SMYTH, H.F. 1964. Effects of repeated inhalation of vapors of industrial solvents on animal behavior. Am. Ind. Hyg. Assoc. J. 25:369-375

KAGAN, E. 1924. Arch Hyg. 94:41 (As cited in Krasavage et al. 1981)

KRASAVAGE, W.J., O'DONOGHUE, J.L., and DIVENCENZO, C.D. 1981. Ketones. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B

NELSON, K.W., EGE, J.F., ROSS, N., WOODMAN, L.E., and SILVERMAN, L. 1943. Sensory response to industrial solvent vapors. J. Ind. Hyg. Toxicol. 25:282-285

Aluminum

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulation; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

GOYER, R.A. 1986. Toxic effects of metals. In Doull, J. Klaassen, C.D., and Amdur, M.D., eds. Casarett and Doull's Toxicology. 3rd ed. MacMillan Publishing Co., New York

NATIONAL ACADEMY OF SCIENCE (NAS). 1982. Drinking Water and Health. 4 vols. National Academy Press, Washington, D.C.

Arsenic

CHEN, C., CHUANG, Y., YOU, S., LIN, T., and WU, H. 1986. A retrospective study on malignant neoplasms of bladder, lung, and liver in blackfoot disease endemic area in Taiwan. Br. J. Cancer 53:399-405

COULSON, E.J., REMINGTON, R.E., and LYNCH, K.M. 1935. Metabolism in the rat of the naturally occurring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10:255-270

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington D.C. EPA 600/8-83-021F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Special Report on Ingested Inorganic Arsenic Skin Cancer: Nutritional Essentiality. Risk Assessment Forum. U.S. Environmental Protection Agency, Washington, D.C. EPA/625/3-87/013F. July 1988

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Health Criteria and Assessment Office, Cincinnati, Ohio.

HOLLAND, R.H., McCALL, M.S., and LANZ, H.C. 1959. A study of inhaled arsenic-74 in man. Cancer Res. 19:1154-1156

RAY-BETTLEY, F., and O'SHEA, J.A. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 92:563-568

TSENG, W.P., CHU, H.M., HOW, S.W., FONG, J.M., LIN, C.S., and YEH, S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463

TSENG, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19:109-119

Barium

BRENNIMAN, G.R., and LEVY, P.S. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J., Calabrese, Ed. Princeton Scientific Publication, Princeton, NJ. p 231-249.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-021

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

GOYER, R.A. 1986. Toxic effects of metals. In Klaasen, C.D., Amdur, M.D., and Doull, J., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 3rd ed. Macmillan Publishing Co., New York. Pp. 623-624

PERRY, H.M., KOPP, S.J., ERLANGER, M.W., and PERRY, E.G. 1983. XVII. Cardiovascular effects of chronic barium ingestion. In Hemphill, D.D., ed. Proceedings of he University of Missouri's 17th Annual Conference of Trace Substances in Environmental Health. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984)

TARASENKO, M., PROMIN, O., and SILAYEV, A. 1977. Barium compounds as industrial poisons (an experimental study). J. Hyg. Epiderm. Microbial. Immunol. 21:361 (As cited in EPA 1984)

WONES, R.G., STADLER, B.L., and FROHMAN, L.A. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ Health Perspective. 85:1-13.

Bromodichloromethane

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Trihalogenated Methanes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Emergency and Remedial Response, Washington, DC. ECAO-CIN-H083.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis studies of Bromodichloromethane in F344/N rats and B6C3F1 mice (Gavage studies). NTP Tech Report Ser No. 321. NIH Publ No. 87-2537.

Bromoform

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR) 1989. Toxicological Profile for Bromoform and Chlorodibromomethane. Prepared by Life Systems for ATSDR, U.S. Public Health Service.

BOWMAN, F., BORZELLECA, J.R., and MUNSON, A.E. 1978. The toxicity of some halomethanes in mice. Toxicol. Appl. Pharmacol. 44(1):213-216.

CHU, I., VILLENEUVE, D.C., SECOURS, V.E., BECKING, G.C., and VALLI, V.E. 1982. Trihalomethanes: II. Reversibility of toxicological changes produced by chloroform, bromodichloromethane, chlorodibromomethane and bromoform in rats. J Environ Sci Health [B] 17(3):225-40.

DYKAN, V.A. 1962. Changes in liver and kidney functions due to methylene bromide and bromoform. Nauchn Tr Ukr Nauchn-Issled Inst Gigieny Truda i Profzabolevanii 29:82-90. (Chem Abstracts 60:8541d).

DYKAN, V.A. 1964. Problems on toxicology, clinical practice, and work hygiene in the production of bromine-organic compounds. Gigiena 5b:100-103. (Chem Abstracts 63:154299).

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio

MINK, F.L., BROWN, T.J., and RICKABAUGH, J. 1986. Absorption, distribution, and excretion of 14C-trihalomethanes in mice and rats. Bull Environ Contam Toxicol. Nov; 37(5):752-8.

NATIONAL TOXICOLOGY PROGRAM (NTP) 1980. (as cited in EPA 1990).

NATIONAL TOXICOLOGY PROGRAM (NTP) 1988. Toxicology and carcinogenesis studies of tribromomethane (bromoform) (CAS No. 75-25-2) in F344/N rats and B6C3F1 mice (gavage studies). Technical report series no. 350. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health, National Toxicology Program. NIH Publication No. 88-2805.

SAX, N.I., and LEWIS, R.J. eds. 1989. Dangerous Properties of Industrial Materials. 7th ed. New York, NY: Van Nostrand Reinhold Company. p. 568.

Cadmium

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (CDHS). 1986. Report to the Scientific Review Panel on Cadmium. Part B. Health Effects of Cadmium. Revised. Prepared by the Epidemiological Studies and Surveillance Section, Berkeley, California. September 19, 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria Document for Cadmium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-025

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Cadmium. Final Draft. Office of Drinking Water, Washington, D.C. April 1985. PB86-117934

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, Washington, D.C. June 1985. EPA 600/8-83-025F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Cadmium. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

FRIBERG, L., PISCATOR, M., NORDBERG, G.F., and KJELLSTROM, T. 1974. Cadmium in the Environment. 2nd ed. CRC Press, Inc., Boca Raton, Florida

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. cadmium production workers——an update. JNCI 74:325-333

Calcium

HAYNES, R.C. and MURAD, F. 1985. Agents affecting Calcification: Calcium, parathyroid hormone, calcitonin, vitamin D, and other compounds. in Gilman AG., Goodman LS., Rall TW., and Murad F, eds. The Pharmacological Basis of Therapeutics. MacMillan Publishing Co., New York, p. 1517-1643.

HEGSTED, D.M. 1957. Calcium requirements. Nutrition Review. 15:257-258 (As cited in NRC 1980)

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Washington DC. National Academy Press. Vol. 3. 269-270.

PEACH, M.J. 1975. Cations: Calcium, Magnesium, Barium, Lithium and Ammonium. in Goodman LS., and Gilman A., eds. The Pharmacological Basis of Therapeutics. 5th ed. MacMillan Publishing Co. Inc. New York.

Carbon Disulfide

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1990. Toxicological profile for Carbon Disulfide. Draft for Public Comment. October 1990.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

ANDREWS, L.S. and SNYDER, R. 1986. Toxic Effects of Solvents and Vapors. In Casarett and Doull's Toxicology. Klaassen C.D., Amdur M.O., and Doull J., eds. 3rd ed. MacMillian Publishing Co., New York. pp. 348

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIR, R.W. 1981. Testing of selected work place chemicals for teratogenic potential. Scand. J. Work Environ. Health 7(Suppl. 4):66-75

PRICE, et al. 1984. Teratologic Evaluation of Carbon Disulfide Administered to Rabbits on GEstation Days 6-19. NTIS PB84-192350

SAX, N.I. 1984. Dangerous Properties of Industrial Materials. Van Nostrand Reinhold Co., New York

TABACOVA, S., B. Nikiforov, L. Balabaeva. 1983. Carbon disulfide intrauterine sensitization. J. Appl. Toxicol. 3:223-229

Chloroform

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

HEYWOOD, R., SORTWELL, R.J. NOEL, P.R.B., et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2:835-851

JORGENSON, T.A., MEIERHENRY, E.F., RUSHBROOK, C.J., et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. Fund. Appl. Toxicol. 5:760-769

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. 0. Bethesda, M.D. DHEW (NIH) Publication No. 76

SCHWETZ, B.A., LEONG, B.K.J., GEHRING, P.J. 1974. Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol. 28:442-451

Chromium

DIAZ-MAYANS, J., LABORDA, R., and NUNEZ, A. 1986. Hexavalent chromium effects on motor activity and some metabolic aspects of Wistar albino rats. Comp Biochem Physiol 83(1):191-195.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office, Research Triangle Park, N.C. EPA 600/8-83-014F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Draft Health Advisory for Chromium. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

IVANKOVIC, S., and PREUSSMAN, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Fd. Cosmet. Toxicol. 13:347-351

LINDBERG, E. and G. HEDENSTIERNA. 1983. Chrome plating: Symptoms, findings in the upper airways, and effects on lung function. Arch. Environ. Health. 38:367-374.

MANCUSO, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Canada

MACKENZIE, R.D., BYERRUM, R.V., DECKER, C.F., HOPPERT, C.A., and LONGHAM, F.L. 1958. Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch. Ind. Health 18:232-234

Cobalt

GILMAN, J.P.W. 1962. Metal carcinogenesis: II. A study on carcinogenic activity of cobalt, copper, iron, and nickel compounds. Cancer Res. 22:158-165

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillian Publishing Co., New York

HEATH, J.C. 1960. The histogenesis of malignant tumors induced by cobalt in the rat. Br. J. Cancer 14:478-482

LEONARD, P., GERBER, G.B., JACQUET, P., and LAUWERYS, R.R. 1984. Mutagenicity, Carcinogenicity and Teratogenicity of Industrially Used Metals. In Kirsch-Volders, M., ed. Mutagenicity, Carcinogenicity and Teratogenicity of Indusrial Pollutants. Plenum Press, New York. Pp. 59-126

NATIONAL RESEARCH COUNCIL (NRC). 1977 Drinking Water and Health. National Academy of Sciences, Washington, D.C.

SHEPARD, T.H. 1986. Catalog of Teratogenic Agents. 5th ed. Johns Hopkins University Press, Baltimore. P. 380

STOKINGER, H.E. 1981. The Metals. In Clayton, C.P., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2A

Copper

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Copper. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA/540-1-86-025

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office, Cincinnati, Ohio, for the Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

Dibromochloromethane

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1989. Toxicological Profile for Bromoform and Chlorodibomomethane. Prepared by Life Systems for ATSDR, U.S. Public Health Service.

BALSTER, R.L. and BORZELLECA, J.F. 1982. Behavioral toxicity of trihalomethan contaminants of drinking water in mice. Environ Health Perspect 46:127-136.

BOWMAN, F., BORZELLECA, J.R. and MUNSON, A.E. 1978. The toxicity of some halomethanes in mice. Toxicol Appl Pharmacol. 44(1):213-126.

CONDIE, L.W., SMALLWOOD, C.L. and LAURIE, R.D. 1983. Comparative renal and hepatotoxicity of halomethanes: Bromodichloromethane, bromoform, chloroform, dibromomethane and methylene chloride. Drug Chem Toxicol 6:562-578.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criterial and Assessment Office. Cincinnati, Ohio.

MINK, F.L., BROWN, J.T. and RICKABAUGH, J. 1986. Absorption, distribution, and excretion of 14C-trihalomethanes in mice and rats. Bull Environ Contam Toxicol 37:752-758.

MUNSON, A.E., SAIN, L.E., SANDERS, V.M., et al. 1982. Toxicology of organ drinking water contaminants: Trichloromethane, bromodichloromethane, dibromochloromethane, and tribromomethane. Environ Health Perspect 46:117-126.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1985. Toxicology and carcinogenesis studies of chlorodibromomethane in F344/N rats and B6C3F1 mice (gavage studies). Technical report series no. 350. Research Triangle Park, NC; U.S. Department of Health and Human Services, Public Health Service, National Institute of Health, National Toxicology Program. NIH Publication No. 88-2805.

RUDDICK, J.A., VILLENEUVE, D.C., CHU, I., et al. 1983. A teratological assessment of four trihalomethanes in the rat. Environ Sci Health B18:333-349.

Diethylphthalate

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. AGCIH, Cincinnati, Ohio

BROWN, D., BUTTERWORTH, K.R., GAUNT, I.F., GRASSO, P., and GANGOLLI, S. 1978. Short-term toxicity study of diethyl phthalate in the rat. Food Cosmet. Toxicol. 16:415-422

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Verified Reference Doses (RfDs) of the USEPA. The ADI Work Group of the Risk Assessment Forum. Cincinnati, Ohio. January 1986. ECAO-CIN-475

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1986. Data base. Registry of Toxic Effects of Chemical Substances. Washington, D.C.

SEED, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45:111-114

Dinitrobenzenes

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENIST (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices 5th ed. ACGIH Inc. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria Assessment Office, Cincinnati, Ohio

CODY, T.E., WINTHERUP, S., HASTINGS, L., STEMMER, K., and CHRISTIAN, R.T. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health 7(5):829-847

BEARD, R. and NOE, J.T. 1981. Aromatic Nitro and Amino Compounds. In Clayton, G.D., and Clayton, P.B. eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2A

2,4-Dinitrotoluene

BERMUDEZ, E., TILLERY, D. and BUTTERWORTH, B.E. 1979. The effect of 2,4-Diaminotoluene and isomers of dinitrotoluene on unscheduled DNA synthesis in primary rat hepatocytes. Environ Mutagen. 1:391-398.

COUCH, D.B., ALLEN, P.F. and ABERNATHY, D.J. 1981. The mutagenicity of dinitrotoluenes in Salmonella typhimurium. Mut Res. 90:373-383.

ELLIS, H.V., III et al. 1976. Subacute toxicity of 2,4-dinitrotoluene and 2,6-dinitrotoluene. Toxicol Appl Pharmacol 37:116 (As cited in EPA 1980).

ELLIS, H.V., HAGEUSEN, S.H., JODGSON, J.R. et al. 1979. Mammalian toxicity of Munitions Compounds. Phase III. Effects of Lifetime Exposure. Part I. 2,4-Dinitrotoluene. NTIS AD-AO77692. p. 281.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Dinitrotoluene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-045.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio

ETNIER, E.L. 1987. Water Quality Criteria for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene. Final Report. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

KEY, M.M. et al. (eds). ,1977. In: Occupational Diseases: A guide to their recognition. U.S. Dep Health Education and Welfare. U.S. Government Printing Office, Washington, D.C. p. 278

LAYTON, D.W., MALLON, B.J., ROSENBLATT, D.H. and SMALL M.J. 1987. Deriving Allowable Daily Intakes for Systemic Toxicants Lacking Chronic Toxicity Data. Regul. Toxicol. Pharmacol. 7:96-112.

LINCH, A.L. 1974. Biological monitoring for industrial exposure to cyanotic aromatic nitro and amino compounds. Am Ind Hyg Assoc Jour 35:426

MIRSALIS, J.C. and BUTTERWORTH, B.E. 1982. Induction of unscheduled DNA synthesis in rat hepatocytes following in vivo treatment with Dinitrotoluenes. Carcinogenesis. 3(3):241-245.

NATIONAL CANCER INSTITUTE. (NCI). 1978. Bioassay of 2,4-Dinitrotoluene for possible carcinogenicity. CAS No. 121-14-2. U.S. Department of Health, Education, and Welfare, Carcinogenesis Testing Program, Bethesda, Maryland. NCI-CG-TR-54. DHEW Publication No. (NIH) 78-1360.

PROCTOR, N.H. and HUGHES, J.P. 1978. Chemical hazards of the Workplace.J.B. Lippincott Co., Philadelphia/Toronto

RICHERT, D.E., BUTTERWORTH, B.E. and POPP, J.A. 1984. Dinitrotoluene: acute toxicity, oncogenicity, genotoxicity and metabolism. CRC Critical Reviews in Toxicology. 13(3).

RYON, M.G., PAL, B.C., TALMAGE, S.S. and ROSS, R.H. 1984. Database Assessment of the Health and Environmental Effects of Munitions Production Waste Products. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

SAX, N. and LEWIS, R. 1989. Dangerous properties of industrial materials Vol III. Van Nostrand Reinhold, New York.

TOKIWA, H., NAKAGAWA, R. and OHNISHI, Y. 1981. Mutagenic assay of aromatic nitro compounds with Salmonella typhimurium. Mut Res. 91:321-325.

WORKING, P.K. and BUTTERWORTH, B.E. 1984. An assay to detect chemically induced DNA repair in rats spermatocytes. Environ Mutagen 6:273-286.

2,6-Dinitrotoluene

ELLIS, H.V., HAGEUSEN, S.H. HODGSON, J.R., et al. 1979. Mammalian Toxicity of Munition Compounds. Phase III. Effects of Lifetime Exposure. Part I. 2,4-Dinitrotoluene. NTIS AD A077692. 281pp.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990.

ETNIER, E.L. 1987. Water Quality Criteria for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene. Final Report. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

HAMBLIN, D.O. 1963. Industrial Hygiene and Toxicology, 2nd ed. Vol. II, pp. 2105-2127. Interscience, New York.

LAYTON, D.W., MALLON, B.J., ROSENBLATT, D.H. and SMALL M.J. 1987. Deriving Allowable Daily Intakes for Systemic Toxicants Lacking Chronic Toxicity Data. Regul. Toxicol. Pharmacol. 7:96-112.

LEE, C.C., DILLEY, J.V., HODGSON, J.R., HELTON, D.O., WIEGAND, W.J., ROBERTS, D.N., ANDERSEN, B.S., HALFPAP, L.M., KURTZ, L.D. and WEST, N. 1975. Mammilian toxicity of munition compounds. Phase I: Acute Oral Toxicity, Primary Skin and Eye Irritation, Dermal Sensitization, and Disposition and Metabolism. AD B011 150. Midwest Research Inst., Kansas City, MO. DAMD17-74-4073.

LEE et al. 1976 (as cited in Etnier 1987--full reference not provided)

LEONARD, T.B., GRAICHEN, M.E. and POPP, J.A. 1987. Dinitrotoluene isomer-specific hepatocarcinogenesis in Fischer-344 rats. J. Nat. Cancer Inst.

LEVINE, R.J., ANDJELKOVICH, D.A., KERSTETER, S.L., ARP, E.W., BALOGH, S.A., BLUNDEN, P.B. and STANLEY, J.S. 1986. Heart disease in workers exposed to dinitrotoluene. J. Occup. Me. 28(9):811-816.

SIMMON, V.F., KAUHANEN, K. and TARDIFF, R.G. 1977. Mutagenic activity of chemicals identified in drinking water. In D. Scott, B.A. Bridges, and F.H. Sobels, eds. Progress in Genetic Toxicology. pp. 249-258. Elsevier, North Holland Biomedical Press, Amsterdam.

SPANGGORD, R.J., MORTELEMANS, K.E., GRIFFIN, A.F. and SIMMON, V.F. 1982. Mutagenicity in Salmonella typhimurium and structure-activity relationships of wastewater components emanating from the manufacture of trinitrotoluene. Environ Mutagen. 4(2):163-179.

TOKIWA, H., NAKAGAWA, R. and OHNISHI, Y. 1981. Mutagenic assay of aromatic nitro compounds with Salmonella-typhimurium. Mutat. Res. 91(4-5):321-326.

VON OETTINGEN, W.F. 1941. The Aromatic Amino and Nitro Compounds, Their Toxicity and Potential Dangers. pp. 108-111. U.S. Public Health Service Bull. No 271.

Di-n-octylphthalate

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment summary Tables. Prepared by the Office of Health and Envrionmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. Registry of Toxic Effects of Chemical Substances Data Base. Washington, D.C. July 1985

NATIONAL TOXICOLOGY PROGRAM AND THE INTERAGENCY REGULATORY LIAISON GROUP (NTP/IRLG). 1982. The Conference on Phthalates. Environ. Health Perspect. 45:1-153

PIEKACZ, H. 1971. Effect of dioctyl and dibutyl phthalates on the organism of rats after oral administration in prolonged experiment. II. Subacute and chronic toxicity. Rocz. Panstw. Zakl. Hig. 22(3):295-307.

Ethylbenzene

ANDREW, F.D., R.L. BUSCHBORM, W.C. CANNON, R.A. MILLER, L.F. MONTGOMERY, D.W. PHELPS, ET AL. 1981. Teratologic assessment of ethylbenzene and 2-ethyxyethanol. Battelle Pacific Northwest Laboratory. Richland, WA. PB 83-208074., 108.

BARDODEJ, Z., and BARDODEJOVA, E. 1970. Biotransformation of ethylbenzene, styrene, and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31:206-209

CHIN, B.H., McKELVEY, J.A., TYLER, T.R., CALISTI, L.J., KOZBELT, S.J., and SULLIVAN, L.J. 1980. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methyl ethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24:477-483

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Ethylbenzene. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7:66-75

WOLF, M.A., ROWE, V.K., McCOLLISTER, D.D., HOLLINGSWORTH, R.L., and OYEN, F. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387-398

bis(2-Ethylhexyl)phthalate

CARPENTER, C.P., WEIL, C.S., and SMYTH, H.F. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs, and dogs. Arch. Indust. Hyg. Occup. Med. 8:219-226

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 40/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of Di(2-ethylhexyl)phthalate in F344 Rats and B6C3F$_1$ Mice. Feed Study. NTP Technical Report Series No. 217, U.S. Department of Health and Human Services. NIH Publication No. 82-1773. NTP-80-37

HMX

DEPARTMENT OF DEFENSE (DOD). 1977. As cited in EPA 1990.

DEPARTMENT OF DEFENSE (DOD). 1985. AD-A171 601. Washington, D.C. As cited in EPA 1990.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Health Advisory on Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

Iron

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Iron. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H054 (Final Draft)

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Safe Drinking Water Committee. National Academy Press, Washington, D.C. Vol. 3, 415 pp

VENUGOPAL, B. and LUCKEY, T.D. 1978. Metal Toxicity in Mammals. In Chemical Toxicity of Metals and Metalloids. Plenum Press, New York

Lead

CENTERS FOR DISEASE CONTROL (CDC). 1985. Preventing Lead Poisoning in Young Children. U.S. Department of Health and Human Services, Atlanta, Georgia

DUGGAN, M.J. 1983. The uptake and excretion of lead by young children. Arch. Env. Health 38147:246-247

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Lead. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-055

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Air Quality Criteria for Lead. Office of Research and Development, Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. June 1986. EPA 600/8-83/023cF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Drinking water regulations maximum contaminant levels goals and national primary drinking water regulations for lead and copper; proposed rule. Fed. Reg. 53:31516-31578 (August 18, 1988)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation. Air Quality Management Division, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. MacMillan Publishing Co., New York. Pp. 421-428

HARLEY, N.H., and KNEIP, T.H. 1985. An Integrated Metabolic Model for Lead in Humans of All Ages. Final report to U.S. Environmental Protection Agency. New York University School of Medicine, Department of Environmental Medicine. January 30, 1985. Contract No. B44899

Magnesium

GOODMAN, L.S. and GILMAN, A.G. EDS. 1985. The Pharmacological Basis of Therapeutics. 7th ed. MacMillan Publishing Co., New York. Pp. 874-878

GOYER, R.A. 1986. Toxic effects of Metals. In Doull, J., Klaassen, C.D., and Amdur, M.D., eds. Casarett and Doull, Toxicology. 3rd ed. MacMillan Publishing Co., New York. Pp. 624-625.

NATIONAL FIRE PROTECTION ASSOCIATION. 1978. Fire Protection Guide on Hazardous Materials. 7th ed. Boston, MA. Pp. 49-81

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. National Institute for Occupational Safety and Health (NIOSH). U.S. Government Printing Office, Washington, D.C.

SAX, N.I. 1984. Dangerous Properties of Industrial Materials. 6th ed. Van Nostrand Reinhold, New York. Pp 1720-1721

STOKINGER, H.E. 1981. The Metals. In Clayton, G., and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley Sons, New York. Vol. 2A. Pp. 1744-1749

Manganese

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. August 1984. EPA 600/8-83-013F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Manganese (and compounds). Environmental Criteria and Assessment Office, Washington, D.C. EPA 540/1-86-057

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washignton, D.C. September 1990

KAWAMURA, R., IKUTA, H., FUKUZUMI, S., et al. 1941. Intoxication by manganese in well water. Kitasato Arch. Exp. Med. 18:145-149

LAI, J.C.K., LEUNG, T.K.C., and LIM, L. 1982 Activities of the mitochondrial NAD-linked isocitric dehydrogenase in different regions of the rat brain. Changes in aging and the effect of chronic manganese chloride administration. Gerontology 28:81-85

LEUNG, T.K.C., LAI, J.C.K., and LIM, L. 1981. The regional distribution of monoamine oxidase activities towards different substrates: Effects in rat brain of chronic administration of manganese chloride and of aging. J. Neurochem. 36:2037-2043

NRC (NATIONAL RESEARCH COUNCIL). 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, D.C. 230-235.

SARIC, M., MARKICEVIC, S., and HRUSTIC, O. 1977. Occupational exposure to manganese. Br. J. Ind. Med. 34:114-118

SCHROEDER, H.A., BALASSA, D.D., AND TIPTON, I.H. 1966. Essential trace metals in man: Manganese, a study in homeostasis. J. Chron. Dis. 19:545-571.

WHO (WORLD HEALTH ORGANIZATION). 1973. Trace elements in human nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. 34-36.

Mercury

BERNAUDIN, J.F., DRUET, E., DRUET, P. and MASSE R. 1981. Inhalation or ingestion of organic or inorganic mercurials produces auto-immune disease in rats. Clin. Immunol. Immunopathol. 20: 488-494.

DRUET, P., DRUET, E., POTDEVIN, F. and SAPIN, C. 1978. Immune type glomerulonephritis induced by $HgCl_2$ in the brown Norway rat. Ann. Immunol. 129C:777-792

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria Document for Mercury. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Water Regulation and Standards, Washington, D.C. EPA 440/5-80-058. NTIS PB 81-117699

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Mercury. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-042

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Resposne, Office of Emergency and Remedial Response, Washington, D.C. September 1990

FAWER, R.F., DERIBAUPIERRE, B., GUILLEMIN, M.P., BERODE, M. and LOBE, M. 1983. Measurement of hand tremor induced by industrial exposure to metalic mercury. J. Ind Med. 40: 204-208.

FITZHUGH, O.G., NELSON, A.A., LAUG, E.P., and KUNZE, F.M. 1950. Chronic oral toxicities of mercury-phenyl and mercuric salts. Arch. Ind. Hyg. Occup. Med 2:433-441

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. Pp. 421-428

LEONARD, A., GERBER, G.B., JACQUET, P., and LAUWERYS, R.R. 1984. Mutagenicity, carcinogenicity, and teratogenicity of industrially used metals. In Kirsch-Volders, M., ed. Mutagenicity, Carcinogenicity and Teratogenicity of Industrial Pollutants. Plenum Press, New York. Pp. 59-126

PIIKIVI, L. 1989. Cardiovascular reflexes and low long-term exposure to mercury vapor. Int Arch Occup Environ Health 61:391-395.

PIIKIVI, L. and HANNINEN, H. 1989. Subjective symptoms and psychlogical performance of chlorine-alkali workers. Scand J Work Environ Health. 15:69-74.

PIIKIVI, L. and TOLONEN, V. 1989. EEG findings in chlro-alkali workers subjected to low long-term exposure to mercury vapor. Br J. Ind MEd. 46:370-375.

RAHOLA, T., HATTULA, T., KORLAINEN, A. and MIETTINEN, J.K. 1971. The biological halftime of inorganic mercury (Hg^{2+}) in man. Scand. J. Clin. Invest. 27(suppl. 116):77 (Abstract)

TASK GROUP ON METAL ACCUMULATION. 1973. Accumulation of toxic metals with special reference to their absorption, excretion and biological halftimes. Environ. Phys. Biochem. 3:65-67

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health Criteria, Mercury. Geneva

Nickel

AMBROSE, A.M., LARSON, P.S., BORZELLECA, J.R., and HENNIGAR, G.R. 1976. Long-term toxicologic assessment of nickel in dogs and rats. J. Food Sci. Technol. 13:181-187

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health Assessment Document for Nickel and Nickel compounds. Office of Health and Environmental Assessment, Research Triangle Park, North Carolina. EPA 600/8-83-012FF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Nickel. Office for Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Resposne, Office of Emergency and Remedial Response, Washington, D.C. September 1990

Nitrobenzene

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1984. Ninety-day Inhalation Study of Nitrobenzene in F344 Rats, CD Rats, and B6C3F1 Mice with cover letter dated 6/24/84 and EPA response dated 8/6/84. Unpublished study. FYI-OTS-0784-0333

DORIGAN, J. and HUSHON, J. 1976. Air Pollution Assessment of Nitrobenzene. U.S. Environmental Protection Agency, Washington, DC

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Nitrobenzene. Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-061

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

PIOTROWSKI, J. 1967. Further investigations on the evaluation of exposure to nitrobenzene. Br. J. Ind. Med. 24:60

Potassium

GOODMAN AND GILMAN. 1985. The Pharmacological Basis of Therapeutics. Gilman A., Goodman L., Rall, T., Murad F. eds. 7th. ed. MacMillan Publishing Co. New York. p. 866-874.

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Volume 3. Safe Drinking Committee, Washington, D.C. 415 pages.

WANDS, R.C. 1981. Alkaline Materials. In Clayton G. and Clayton F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pg. 3054-3055.

RDX

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Health Advisory for Hexahydro-1,3,5-Trinitro-1,3,5-Triazine (RDX). Criteria and Standards Division, Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

GOSSELIN, R.E., SMITH, R.P, HODGE, H.C. and BRADDOCK, J.E. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins, Baltimore.

HART, E.R. 1977. Two-year feeding study in rats. AD A040161. Litton Bionetics, Inc., Kensington, MD. Office of Naval Research, Contract No. N00014-73-C-0162.

HATHAWAY, J.A. ET AL. 1977. J. Occup. Med. 19:269-272. As cited in ACGIH.

KAPLAN, A.S., BERGHOUT, C.F. and PECZENIK, A. 1965. Human intoxication from RDX. Arch. Environ. Health 10:877-883.

LEVINE, B.S., FUREDI, E.M. RAC, V.S., GORDON, D.E. and LISH, P.M. 1983. Determination of the chronic mammalian toxicological effects of RDX: Twenty-four month chronic toxicity/carcinogenicity study of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the Fischer 344 rat. Phase V. Vol.1. AD A160774. Chicago, IL: IIT Research Institute. U.S. Army Medical Research and Development Command, Contract No. DAMD17-79-C-9161.

LISH, P.M., LEVINE, B.S., FUREDI, E.M., SAGARTZ, E.M. and RAC, V.S. 1984. Determination of the chronic mammalian toxicological effects of RDX: Twenty-four month chronic toxicity/carcinogenicity study of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 hybrid mouse. Phase VI. Vol.1. Chicago, IL: IIT Research Institute. U.S. Army Medical Research and Development Command, Contract No. DAMD17-C-9161.

SUNDERMAN, F. ET AL. 1944. National Defense Research Comm., Office of Scientific Research and Development. NDRC Contract OEM sr-96a.

VON OETTINGEN, W.F., DONAHUE, D.D., YAGODA, H., ET AL. 1949. J. Ind. Hyg. Tot. 31:21. As cited in ACGIH.

Silver

BLUMBERG, H. and CAREY, T.N. 1934. Argyremia: Detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. J. Am. Med. Assoc. 103:1521-1524

EAST, B.W., BODDY, K., WILLIAMS, E.D., MACINTYRE, D. and McLAY, A.L.C. 1980. Silver retention, total body silver and tissue silver concentrations in argyria associated with exposure to an anti-smoking remedy containing silver acetate. Clin. Exp. Dermatol. 5:305-311

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Silver. Environmental Criteria and Assessment Office, Cincinnati, Ohio. PB 86-118288

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

GAUL, L.E. and STAUD, A.H. 1935. Clinical spectroscopy. Seventy cases of generalized argyrosis following organic and colloidal silver medication. J. Am. Med. Assoc. 104:1387-1390

HILL, W.R. and PILLSBURY, D.M. 1939. Argyria, the Pharmacology of Silver. Williams and Wilkins Co., Baltimore, Maryland.

Sodium

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rulemaking. Fed. Reg. 50:46936-47025.

NATIONAL ACADEMY OF SCIENCES (NAS). 1977. Drinking Water and Health. Safe Drinking Water Committee, Washington, D.C. p. 400-411

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1987. Registry of Toxic Effects of Chemical Substances. Data Base. Washington D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Volume 3. Safe Drinking Water Committee, Washington, D.C. 415 pages.

NISHIMOTO, H. and MIYAMOTO, S. 1969. Teratogenic effects of sodium chloride in mice. Acta Anat. 74:121-124.

WANDS, RC. 1981. Alkaline Materials. In Clayton G. and Clayton F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pg. 3056-3057.

1,1,2,2-Tetrachloroethane

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,1,2,2-Tetrachloroethane. Environmental Criteria Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-032

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

HORIUCHI, K., HORIGUCHI, K., KADOWAKI, K., and ARATAKE, K. 1962. Studies on the industrial tetrachloroethane poisoning. Osaka City Med. J. 8:29-38

NAVROTSKIY, V.K., KASHIN, L.M., KULINSKAYA, I.L., ET AL. 1971. Comparative assessment of the toxicity of a number of industrial poisons when inhaled in low concentrations for prolonged periods. Trudy S'ezda Gigenistov Ukranisoi 8:224-226 (Russian)

NATIONAL CANCER INSTITUTE (NCI). 1978. Biassay of 1,1,2,2-Tetrachloroethane for Possible Carcinogenicity. NCJ Tech. Rep. Ser. No. 27

SCHMIDT, P., BINNEWIES, S., GOHLKE, R., and ROTHE, R. 1972. Subacute action of low concentrations of chlorinated ethanes on rats with and without additional ethanol treatment. I. Biochemical and toxicometric aspects, especially results in subacute and chronic toxicity studies with 1,1,2,2-tetrachloroethane. Int. Arch. Arbeitsmed. 30:283-298 (German)

Tetryl

HAMILTON, A. and HARDY, H. 1974. Industrial Toxicology, 3rd. ed., p. 308. Pub. Sciences Group, Inc., Acton, MA.

HARDY, W.L. and MALOOF, C.C. 1950. Evidence of systemic effect of tetryl. AMA Arch. Ind. Hyg. Occup. Med. 1:545.

SCHWARTZ, L. 1942. Industrial dermatitis in our war industries. Ind. Med. 11(10):457.

WELLS, H.G., LEWIS, J.H., SANSUM, W.D. ET AL. 1920. Observations on the toxicity of teranitromethyl aniline (Tetryl), tetranitroxylene (T.N.X.), tetranitroaniline (T.N.A.), dinitrodichlorobenzene, parazol, and metanitroaniline. J. Ind. Hyg. 2:247.

WHONG, W.Z., SPECINER, N.D. and EDWARDS, G.S. 1980. Mutagenic activity of tetryl, a nitroaromatic explosive, in three microbial test systems. Toxicology Letters 5:11-17.

WITKOWSKI, L.J. FISHER, C.N. and MURDOCK, H.D. 1942. Industrial illness due to tetryl. JAMA 119(17):1406.

Toluene

ANDERSON, I., LUNDQVIST, G.R., MOLHAVE, L., PEDERSEN, O.F., PROCTOR, D.F., VEATH, M. and WYON, P.P. 1983. Human response to controlled levels of toluene in six-hour exposure. Scand. J. Work Environ Health. 9:405-418.

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1980. A Twenty-Four Month Inhalation Toxicology Study in Fischer 344 Rats Exposed to Atmospheric Toluene. Executive Summary and Data Tables. October 15, 1980

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Toluene. Final Draft. Office of Drinking Water, Washington, D.C. March 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Toluene. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

HANNINEN, H., ESKELININ, L., HUSMAN, K. and NURMINEEN, M. 1976. Behavioral effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health 2:240-255 (As cited in EPA 1987)

HUDAK, A. and UNGVARY, G. 1978. Embryotoxic effects of benzene and its methyl derivatives: Toluene, xylene. Toxicology 11:55-63

KIMURA, E.T., EBERT, D.M. and DODGE, P.W. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19:699-704

NAWROT, P.S. and STAPLES, R.E. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A

NTP (National Toxicology Program). 1989. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F$_1$ mice. Technical Report Series No. 371. Research Triangle Park, NC.

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942a. The Toxicity and Potential Dangers of Toluene, with Special Reference to its Maximal Permissible Concentration. PHS Publication No. 279. P. 50 (As cited in EPA 1987)

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942b. The toxicity and potential dangers of toluene—preliminary report. J. Am. Med. Assoc. 118:579-584 (As cited in EPA 1987)

WOLF, M.A., ROWE, V.K., MCCOLLISTER, D.D., et al. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387

1,1,2-Trichloroethane

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. AGCIH, Inc., Cincinnati, Ohio

ARENA, J.M. 1979. Poisoning: Toxicology, Symptoms, Treatments. 4th ed., Charles C. Thomas, Publisher, Springfield, Illinois. P. 207

ELOVAARA, E., et al. 1979. Effects of methylene chloride, trichloroethane, trichloroethylene, tetrachloroethylene, and toluene on the development of chick embryos. Toxicology. 12:111

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Health Criteria and Assessment Officer, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. Dept. Health, Educ, Welfare. Pub. No. NCI-CG-TR-74

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health, Volume 1, National Academy Press, Washington, D.C. P. 775-776

SANDERS, V.M., WHITE Jr., K.L., SHOPP Jr., G.M., and MUNSON,A.E. 1985. Humoral and cell mediated immune status of mice exposed to 1,1,2-trichloroethane. Drug Chem. Toxicol 8:357-372

TORKELSON, T.R., AND ROWE, U.K. 1981. Halogenated alphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B., p. 3510-3512

WHITE, K.L., Jr., SANDERS, V.M., BARNES, V.W., SHOPP, G.M., Jr. and MUNSON,A.E. 1985. Toxicology of 1,1,2-trichloroehtane in the mouse. Drug Chem. Toxicol 8: 333-355

WEISBURBER, E.K. 1977. Carcinogenicity studies on halogenated hydrocarbons. Environ. Health Perspect. 21:7-16

Trichloroethylene

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Trichloroethylene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-046

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina. EPA/600/8-82/006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Trichloroethylene. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

FUKUDA, K., TAKEMOTO, K. and TSURUTA, H. 1983. Inhalation carcinogenicity of trichloroethylene in mice and rats. Ind. Health 21:243-254

GREIM, H., BIMBOES, D., EGERT, G., GIGGELMANN, W. and KRAMER, M. 1977. Mutagenicity and chromosomal aberrations as an analytical tool for *in vitro* detection of mammalian enzyme-mediated formation of reactive metabolites. Arch. Toxicol. 39:159

KIMMERLE, G. and EBEN, A. 1973. Metabolism, excretion and toxicology of trichloroethylene after inhalation. 1. Experimental exposure on rat. Arch. Toxicol. 30:115

KJELLSTRAND, P., HOLQUIST, B., ALM, P., KANJE, M., ROMARE, S., JONSSON, I., MANNSON, L. and BJERKEMO, M. 1983. Trichloroethylene: Further studies of the effects on body and organ weights and plasma butyl cholinesterase activity in mice. Acta. Pharmacol. Toxicol. 53:375-384 (As cited in EPA 1985)

MALTONI, C., LEFEMINE, G. and COTTI, G. 1986. Experimental Research on Trichloroethylene. Carcinogenesis Arch. Res. Industrial Carcinogenesis Series. Maltoni, C., Mehlman, M, A., Eds. Vol. V. Princeton Scientific Publishing Co., Inc., Princeton, NJ. p. 393

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264 122

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F$_1$ mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.

STEPHENS, C. 1945. Poisoning by accidental drinking of trichloroethylene. Br. Med. J. 2:218

TORKELSON, T.R. and ROWE, V.K. 1981. Halogenated aliphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B, pp. 3553-3559

Trichlorofluoromethane

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Edition. Cincinnati, Ohio

ADIR, J., BLAKE, D.A. and MERGNER, G.M. 1975. J. Clin. Pharmacol. 15:760

AVIADO, D.M. 1975. Toxicology 3:311

BARRAS, C.E. 1974. Unpublished data. Haskell Laboratory, DuPont Co., Wilmington, DE. October 1974

CLARKE, D.G. and TINSTON, D.J. 1972. Proc. Eur. Soc. Drug Toxicity 13:212

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

FLOWERS, N.C., HAND, R.C. and HORAN, L.G. 1975. Arch. Env. Health. 30:353

HOOD, D.B. 1964. Unpublished data. Haskell Laboratory, DuPont Co., Wilmington, DE. September 1964

JENKINS, L.J., JONES, R.A., COON, R.A. and SIEGEL, J. 1970. Tox. Appl. Pharm. 16:133

LESTER, D. and GREENBERG, L.A. 1950. Arch. Ind. Hyg. Occup. Med. 2:335

MERGNER, G.W., BLAKE, D.A. and HELRICH, M. 1975. Anesthesiology 42:345

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Trichlorofluoromethane for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 106. (Also publ as DHEW (NIH) 78-1356)

PAULET, G., RONCIN, G., and VIDAL, E., et al. 1975. Tox. Apl. Pharm. 34:197

QUEVAUVILLER, A., SCHRENZEL, M. and HUYEN, V.N. 1964. Therapie 19:247

REINHARDT, C.F., AZAR, A. and MAXFIELD, ME., et al. 1971. Arch. Env. Health. 22:265

SCHOLZ, J. 1962. Fortschr. Biol. Aerosolforsch. Jahren 1957-1961. Ber. Aerosol-Kongr. 4:420

SLATER, T.F. 1965. Biochem. Pharmacol. 14:178

TAYLOR, G.J. 1975. Arch. Env. Health. 30:349

TAYLOR, G.J., and DREW, R.T. 1975. Tox. Appl. Pharm. 32:177

1,3,5-Trinitrobenzene

CODY, T.E., WITHERUP, S., HASTINGS, L., STEMMER, K. and CHRISTIAN, R.T. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health 7(5): 829-847.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1983. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health.

2,4,6-Trinitrotoluene

BAILEY, H.W., SPANGGORD, R.J., JAVITZ, H.S. and LIU, D.H.W. 1985. Toxicity of TNT Wastewaters to Aquatic Organisms. Volume III. Chronic Toxicity of LAP Wastewater and 2,4,6-Trinitrotoluene. Final Report. SRI International, Menlo Park, CA. DAMD17-75-C-5056. 80pp.

DILLEY, J.V., TYSON, C.A., SPANGGORD, R.J., SASMORE, D.P., NEWELL, G.W and DACRE, J.C. 1982. Short-term oral toxicity of 2,4,6-trinitrotoluene in mice, rats, and dogs. J. Tox. Environ. Health 9:565-585.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Dinitrotoluene. Criteria and Standards Division, Washington, D.C. EPA 440/5-80-045. NTIS PB 81-117566.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Trinitrotoluene. Health Advisory. Office of Drinking Water, U.S. EPA. Washington, D.C. 20460.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

HARKONEN, H., KARKI, M., LAHTI, A. and SAVOLAINEN, H. 1983. Early equational cataracts in workers exposed to trinitrotoluene. Am. J. Ophth. 95:807-810.

KAGANOV, A.L., FEDYUSHINA, N.A. and BOCHENKOVA, Y.K. 1970. [A study of the nervous system under prolonged exposure to trinitrotoluene.] Gig. Tr. Prof. Zabol. 14: 47-48 (Russian), as cited in Zakhari and Villaume, 1978.

LEE, C-C, DILLEY, J.V., HODGSON, J.R. et al. 1975. Mammalian toxicity of munition compounds: Phase I. Acute oral toxicity, primary skin and eye irritation, dermal sensitization, and disposition and metabolism. Report No. 1, Midwest Research Institute, Project No. 3900-B, Kansas City, MO. DAMD 17-74C-4073. AD-B011 150.

LEVINE, RUSH, BURNS, AND LISH. 1983. Determination of chronic mammalian effects of TNT: 26-Week subchronic oral toxicity of TNT in beagle dogs. Department of Defense. Cameron Station, Alexandria, Virginia. AD-A157-002.

LIU, D.H.W., SPANGGORD, R.J., BAILEY, H.C., JAVITZ, H.S. and JONES, D.C.L. 1983. Toxicity of TNT Wastewaters to Aquatic Organisms. Volume I. Acute Toxicity of LAP Wastewater and 2,4,6-Trinitrotoluene. Final Report. AD A142 144. SRI International, Menlo Park, CA. DAMD17-75-C-5056. 85pp.

NATIONAL RESEARCH COUNCIL. 1982. National Research Council. Evaluation of the health risks of ordinance disposal waste in drinking water. Committee on Toxicology. National Academy Press. Washington, D.C. N00014-80-C-0161. PB83 153635.

RYON, M.G. 1987. Water Quality Criteria for 2,4,6-Trinitrotoluene (TNT). Final Report. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

RYON, M.G., PAL, B.C., TALMAGE, S.S. and ROSS, R.H. 1984. Database Assessment of the Health and Environmental Effects of Munitions Production Waste Products. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

SPANGGORD, R.J., MORTELMANS, K.E., GRIFFIN, A.F. and SIMMON, V.F. 1982. Mutagenicity in *Salmonella typhimurium* and structure-activity relationships of wastewater compounds emanating from the manufacture of trinitrotoluene. Environ. Mutagen. 4: 163-179.

U.S. DOD (DEPARTMENT OF DEFENSE). 1984. Cameron Station, Alexandria, Virginia. AD-A168637.

ZAKHARI A. and VILLAUME, J.E. 1978. A Literature Review - Problem definition studies on selected toxic chemicals. Volume 3 of 8. Occupational health and safety aspects of 2,4,6-trinitrotoluene (TNT). Final Report, Science Information Services Organization, Philadelphia, PA. DAMD 17-77-C-7020, AD-055 683.

Vanadium

BROWNING, E. 1969. Toxicity of Industrial Metal. 2nd ed. Appleton-Century-Crofts, New York

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Health Criteria and Assessment Office, Cincinnati, Ohio

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Vanadium. Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, Washington, D.C.

NATIONAL INSTITUTE OF OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Vanadium. DHEW (NIOSH) Publication No. 77-222

SCHROEDER, J.A., MITCHNER, M. and NASON, A.P. 1970. Zirconium, niobium, antium, antimony, vanadium and lead in rats: Life term studies. J. Nutr. 100(1):59-68

STOKINGER, H.E., WAGNER, W.E., MOUTAIN, J.T., STACKSILL, F.R., DOBROGORSKI, O.J. and KEENAN, R.G. 1953. Unpublished Results. National Institute for Occupational Safety and Health, Division of Occupational Health, Cincinnati, Ohio

Xylenes

BOWERS, D.E., Jr., CANNON, M.S. and JONES, D.H. 1982. Ultrastructural changes in livers of young and aging rats exposed to methylated benzenes. Am. J. Vet. Res. 43:679-683

CARPENTER, C.P., KINKEAD, E.R., GEARY, D.L., Jr., SULLIVAN, L.J. and KING, J.M. 1975. Petroleum hydrocarbon toxicity studies: V. Animal and human response to vapors and mixed xylenes. Toxicol. Appl. Pharmacol. 33:543-558

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Xylenes (Final Draft). Environmental Criteria and Assessment Office, Cincinnati, Ohio. March 1985. ECAO-CIN-416. EPA 600/X-84-185-1

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Xylenes. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1991. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

HAKE, C.L., R.D. STEWARD, A. WU, et al. 1981. Development of a biologic standard for the industrial worker by breath analysis. NIOSH-MCOW-ENUM-XY-77-3. NTIS PB82-152844.

MALTONI, C., CONTI, B., COTTI, G. and BELPOGGI, F. 1985. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7:415-416

MIRKOVA, E., ANTON, G., MIKHAILOVA, A., KHINKOVA, L. and BENCHEV, I.V. 1983. Prenatal toxicity to xylene. J. Hyg. Epidemiol. Microbiol. Immunol. 27:337-343

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis Studies of Xylenes (Mixed) [60% *m*-Xylene; 14% *p*-Xylene; 9% *o*-Xylene; 17% Ethylbenzene] in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies). December 1986. NTP TR 327

SAVOLAINEN, K., RIIHIMAKI, V., SEPPALAINEN, A.M. and LINNOILA, M. 1980. Effects of short-term *m*-xylene exposure and physical exercise on the central nervous system. Int. Arch. Occup. Environ. Health 37:205-217

TATRAI, E., UNGVARY, G., and CSEH, I.R. 1981. The effect of long-term inhalation of *o*-xylene on the liver. Ind. Environ. Xenobiotics Proc. Int. Conf. Pp. 293-300

Zinc

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Zinc (and Compounds). Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-048. September 1984

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. September 1990

HAMMOND, P.B. and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. Pp. 409-467

PORIES, W.J., HENZEL, J.H., ROB, C.G. and STRAIN, W.H. 1967. Acceleration of wound healing in man with zinc sulfate given by mouth. Lancet. 1:121-124

PRASAD, A.S., SCHOOMAKER, E.B., ORTEGA, J., et al. 1975. Zinc deficiency in sickle cell disease. Clin. Chem. 21:582-587

APPENDIX Q

LIST OF SPECIES POTENTIALLY PRESENT AT MAAP

VEGETATION

Common Name	Scientific Name
TREES	
Green ash	*Fraxinus pennsylvanica*
White ash	*Fraxinus americana*
Beech	*Fagus grandifolia*
River birch	*Betula*
Boxelder	*Acer negundo*
E. red cedar	*Juniperus virginiana*
Black cherry	*Prunus serotina*
Cottonwood	*Populus deltoides*
Dogwood	*Cornus florida*
American elm	*Ulmus americana*
Slippery elm	*Ulmus rubra*
Winged elm	*Ulmus alata*
Black gum	*Nyssa sylvatica*
Sweet gum	*Liquidambar styraciflua*
Pignut hickory	*Carya glabra*
Shagbark hickory	*Carya ovata*
Holly	*Ilex opaca*
Black locust	*Acer rubrum*
Black oak	*Quercus velutina*
Blackjack oak	*Quercus marilandica*
Bur oak	*Quercus macrocarpa*
N. red oak	*Quercus rubra*
Pin oak	*Quercus palustris*
Post oak	*Quercus stellata*
Scarlet oak	*Quercus coccinea*
Single oak	*Quercus imbricaria*
S. red oak	*Quercus falcata*
Swamp chestnut oak	*Quercus michauxii*
Water oak	*Quercus nigra*
White oak	*Quercus alba*
Willow oak	*Quercus phellos*
Osage orange	*Maclura pomifera*
Pecan	*Carya illinoensis*
Persimmon	*Diospyros virginiana*
Loblolly pine	*Pinus taeda*
Shortleaf pine	*Pinus echinata*
Virginia pine	*Pinus virginiana*
Yellow poplar	*Liriodendron tulipifera*
Sassafras	*Sassafrass albidum*
Sycamore	*Plantanus occidentalis*
Black walnut	*Juglans nigra*

Common Name	Scientific Name
SHRUBS AND VINES	
Blackberry	*Rubus spp.*
Elderberry	*Sambucus canadensis*
Grape	*Vitis argentifolia*
Honeysuckle	*Lonicera japonica*
Kudzu	*Pueraria lobata*
Sumac	*Rhus spp.*
Trumpet creeper	*Campsis radicans*
Wild plum	*Prunus lanata*
GRASSES AND CLOVERS	
Alfalfa	*Medicago sativa*
Annual lespedeza	*Lespedeza striata & stipulacea*
Barley	*Hordeum vulgare*
Begger-lice	*Desmodium*
Bermuda grass	*Cynodon dactylon*
Broomsedge	*Andropogon virginicus*
Buckthorn plantain	*Plantago lanceolata*
Burclover	*Medicago arabica*
Canada thistle	*Cirsium arvense*
Corn	*Zea mays*
Crabgrass	*Digitaria sanguinalis*
Crimson clover	*Trifolium incarnatum*
Dallis grass	*Pappalsum dilatatum*
Dodder	*Cuscuta spp.*
Goose grass	*Eleusine indica*
Hairy vetch	*Vicia villosa*
Hop clover	*Trifoliumprocumbens*
Horseweed	*Erigeron canadensis*
Jimsonweed	*Datura stramonium*
Johnsongrass	*Sorghum halepense*
Kentucky bluegrass	*Poa pratensis*
Lespedeza bicolor	*Desnodium bicolor*
Lespedeza sericea	*Lespedeza sericea*
Orchard grass	*Dactylis glomerata*
Oxeye daisy	*Chrysanthemum leucanthemum*
Partridgepea	*Chamaecrista spp.*
Pigweed	*Amaranthus spp.*

Common Name	Scientific Name
GRASSES AND CLOVERS (Cont.)	
Pokeweed	*Phytolacca americana*
Purpletop	*Tridens flava*
Quackgrass	*Agropyron repens*
Ragweed	*Ambrosia artemisifolia*
Red clover	*Trifolium pratense*
Red top	*Agristis alba*
Ryegrass	*Lolium multiforum*
Sorghum	*Sorghum vulgare*
Soybeans	*Glycine max*
Sudan grass	*Sorghum volgare sudanense*
Tall Fescue	*Festuca arundinacea*
White clover	*Trifolium pratense*
Wild garlic	*Allium vineale*
Wild mustard	*Brassica kaber*

MAMMALS

Common Name	Scientific Name
Fox squirrel	*Sciurus niger*
Gray squirrel	*Scuirus carolinensis*
Rabbit	*Sylvilagus floridanus*
Raccoon	*Procyon lotor*
Whitetailed deer	*Odocoileus virginianus*
Beaver	*Castor canadensis*
Bobcat	*Lynx rufus*
Gray fox	*Urocyon cinereoargenteus*
Groundhog	*Marmota monax*
Mink	*Mustela vision*
Muskrat	*Ondatra zibethica*
Opossum	*Didelphis marsupialis*
Red fox	*Vulpes fulva*
Skunk	*Mephitis mephitis*
Coyote	*Canis latrans*
River otter	*Riparius otor*

Common Name	Scientific Name
Pied-billed grebe	*Podilymbus podiceps*
Great blue heron	*Ardea herodias*
Green heron	*Butorides virescens*
Little blue heron	*Florida caerulea*
Cattle Egret	*Bubulcus ibis*
Black-crowned night heron	*Nycticorax nycticorax*
Yellow-crowned night heron	*Nyctanassa violacea*
Least Bittern	*Ixobrychus exilis*
Canada goose	*Branta canadensis*
Snow goose	*Chen caerulescens*
Mallard	*Anas platyrhynchos*
Black duck	*Anas rubripes*
Gadwall	*Anas strepera*
Pintail	*Anas acuta*
Green-winged teal	*Anas carolinensis*
Blue-winged teal	*Anas discors*
American wigeon	*Anas americana*
Northern shoveler	*Anas clypeata*
Wood duck	*Aix sponsa*
Redhead	*Aythya americana*
Ring-necked duck	*Aythya collaris*
Lesser Scaup	*Aythya affinis*
Bufflehead	*Bucephala albeola*
Ruddy duck	*Oxyura jamaicensis*
Hooded Merganser	*Lophodytes cucullatus*
Turkey vulture	*Cathartes aura*
Black vulture	*Coragyps atratus*
Sharp-shinned hawk	*Accipeter striatus*
Cooper's hawk	*Accipeter cooperii*
Red-tailed hawk	*Buteo jamaicensis*
Red-shouldered hawk	*Buteo lineatus*
Broad-winged hawk	*Buteo platypterus*
Marsh hawk	*Circus cyaneus*
American kestrel	*Falco sparverius*
Bobwhite	*Colinus virginianus*
Turkey	*Meleagris gallopava*
King rail	*Rallus elegans*
Sora	*Porzana carolina*
American coot	*Fulica americana*
Semipalmated plover	*Charadrius semipalmatus*
Killdeer	*Charadrius vociferus*
American golden plover	*Pluvialis dominica*

Common Name	Scientific Name
Black-bellied plover	*Squatarola squatarola*
American woodcock	*Philohela minor*
Common snipe	*Capella gallinago*
Spotted sandpiper	*Actitis macularia*
Solitary sandpiper	*Tringa solitaria*
Greater yellowlegs	*Tringa melanoleucus*
Lesser yellowlegs	*Tringa flavipes*
Pectoral sandpiper	*Calidris melanotos*
White-rumped sandpiper	*Calidris fuscicollis*
Least sandpiper	*Calidris minutilla*
Dunlin	*Calidris alpina*
Semipalmated sandpiper	*Calidris pusillus*
Rock dove	*Columba livia*
Mourning dove	*Zenaida macroura carolinensis*
Yellow-billed cuckoo	*Coccyzus americanus*
Black-billed cuckoo	*Coccyzus erythropthalmus*
Barn owl	*Tyto alba*
Screech owl	*Otus asis*
Great horned owl	*Bubo virginianus*
Barred owl	*Strix varia*
Chuck-will's-widow	*Caprimulgus carolinensis*
Whip-poor-will	*Caprimulgus vociferus*
Common nighthawk	*Chordeiles minor*
Chimney swift	*Chaetura pelagica*
Ruby-throated hummingbird	*Archilochus colubris*
Belted kingfisher	*Megaceryle alcyon*
Common Flicker	*Colaptes auratus*
Pileated woodpecker	*Dryocopus pileatus*
Red-bellied woodpecker	*Centurus carolinus*
Red-headed woodpecker	*Melanerpes erythrocephalus*
Yellow-bellied sapsucker	*Sphyrapicus varius*
Hairy woodpecker	*Dendrocopos villosus*
Downy woodpecker	*Dendrocopos pubescens*
Eastern kingbird	*Tyrannus tyrannus*
Great crested flychatcher	*Myiarchus crinitus*
Eastern phoebe	*Sayornis phoebe*
Acadian flycatcher	*Empidonax virescens*
Willow flycatcher	*Empidonax traillii*
Eastern wood pewee	*Contopus virens*
Horned lark	*Eremophila alpestris*
Tree swallow	*Iridoprocne bicolor*

BIRDS (Continued)

Common Name	Scientific Name
Bank swallow	*Riparia riparia*
Rough-winged swallow	*Stelgidopteryx ruficollis*
Barn swallow	*Hirundo rustica*
Cliff swallow	*Petrochelidon pyrrhonota*
Purple martin	*Progne subis*
Blue jay	*Cyanocitta cristata*
Common crow	*Corvus brachyrhynchos*
Carolina chickadee	*Parus carolinensis*
Tufted titmouse	*Parus bicolor*
White-breasted nuthatch	*Sitta carolinensis*
Brown creeper	*Certhia familiaris*
House wren	*Troglodytes aldon*
Winter wren	*Troglodytes troglodytes*
Bewicks' wren	*Thryomanes bewickii*
Carolina wren	*Thryothorus ludovicianus*
Northern mockingbird	*Mimus polyglottos*
Gray catbird	*Dumetella carolinensis*
Brown thrasher	*Toxostoma rufum*
American robin	*Turdus migratorius*
Wood thrush	*Hylocichla mustelina*
Hermit thrush	*Catharus guttata*
Swainson's thrush	*Catharus ustulata*
Gray-cheeked thrush	*Catharus minima*
Eastern bluebird	*Sialia sialis*
Blue-gray gnatcatcher	*Polioptila caerulea*
Golden-crowned kinglet	*Regulus satrapa*
Ruby-crowned kinglet	*Regulus calendula*
Water pipit	*Anthus spinoletta*
Cedar waxwing	*Bombycilla cedrorum*
Loggerhead shrike	*Lanius ludovicianus*
Starling	*Sturnus vulgaris*
White-eyed vireo	*Vireo griseus*
Yellow-throated vireo	*Vireo flavifrons*
Solitary vireo	*Vireo solitarius*
Red-eyed vireo	*Vireo olivaceus*
Warbling vireo	*Vireo gilvus*
Black-and-white warbler	*Mniotilta varia*
Prothonotary warbler	*Protonotaria citrea*
Worm-eating warbler	*Helmitheros vermivorus*
Golden-winged warbler	*Vermivora chrysoptera*
Blue-winged warbler	*Vermivora pinus*
Tennessee warbler	*Vermivora peregrina*

Common Name	Scientific Name
Orange-crowned warbler	*Vermivora celata*
Nashville warbler	*Vermivora ruficapilla*
Northern parula	*Parula americana*
Yellow warbler	*Dendroica petechia*
Magnolia warbler	*Dendroica magnolia*
Cape may warbler	*Dendroica tigrina*
Yellow-rumped (Myrtle) warbler	*Dendroica coronata*
Black-throated green warbler	*Dendroica virens*
Cerulean warbler	*Dendroica cerulea*
Yellow-throated warbler	*Dendroica dominica*
Chestnut-sided warbler	*Dendroica pensylvanica*
Bay-breasted warbler	*Dendroica castanea*
Blackpoll	*Dendroica striata*
Pine warbler	*Dendroica pinus*
Prairie warbler	*Dendroica discolor*
Palm warbler	*Dendroica palmarum*
Ovenbird	*Seiurus aurocapillis*
Northern waterthrush	*Seiurus noveboracensis*
Louisiana waterthrush	*Seiurus motacilla*
Kentucky warbler	*Oporornis formosus*
Connecticut warbler	*Oporornis agilis*
Mourning warbler	*Oporornis philadelphia*
Common yellowthroat	*Geothlypis trichas*
Yellow-breasted chat	*Icteria virens*
Hooded warbler	*Wilsonia citrina*
Wilson's warbler	*Wilsonia pusilla*
Canada warbler	*Wilsonia canadensis*
American redstart	*Setophaga ruticilla*
House sparrow	*Passer domesticus*
Bobolink	*Dolichonyx oryzivorus*
Eastern meadowlark	*Sturnella magna*
Red-winged blackbird	*Agelaius phoeniceus*
Orchard oriole	*Icterus spurius*
Northern (Baltimore) oriole	*Icterus galbula*
Rusty blackbird	*Euphagus carolinus*
Brewer's blackbird	*Euphagus cyanocephalus*
Common grackle	*Quiscalus quiscula*
Brown-headed blackbird	*Molothrus ater*
Scarlet tanager	*Piranga olivacea*
Summer tanager	*Piranga rubra*
Cardinal	*Cardinalis cardinalis*
Rose-breasted grosbeak	*Pheucticus ludovicianus*

BIRDS (Continued)

Common Name	Scientific Name
Blue grosbeak	*Guiraca caerules*
Indigo bunting	*Passerina cyanea*
Dickcissel	*Spiza americana*
Purple finch	*Carpodacus purpureus*
American goldfinch	*Spinus tristis*
Ruous-sided towhee	*Pipilo erythrophthalmus*
Savannah sparrow	*Passerculus sandwichensis*
Grasshopper sparrow	*Ammodramus savannarum*
Vesper sparrow	*Poolcetes gramineus*
Dark-eyed junco	*Junco hyemalis*
Tree sparrow	*Spizella arborea*
Chipping sparrow	*Spizella passerina*
Field sparrow	*Spizella pusilla*
White-crowned sparrow	*Zonotrichia leucophyrys*
White-throated sparrow	*Zonotrichia albicollis*
Fox sparrow	*Passerella iliaca*
Swamp sparrow	*Melospiza georgiana*
Song sparrow	*Melospiza melodia*
Quail	*Colinis virgnianus*
Wild turkey	*Meleagris gallopavo*

FISH

Common Name	Scientific Name
Channel catfish	*Ictalurus punctatus*
Largemouth bass	*Micropterus salmodes*
Bluegill	*Lepomis macrochirus*

SOURCES:

MARTIN MARIETTA ALUMINUM SALES, INC. 1978. Environmental Impact Assessment, Milan Army Ammunition Plant, Milan, Tennessee. Prepared for the Department of the Army, August 1, 1978.

MARTIN MARIETTA. 1991. Natural Resources Management Plan for MAAP. Information received from Michael Harris, February 26, 1191.

APPENDIX R

FEDERAL GROUNDWATER CRITERIA

APPENDIX R

APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS) AND OTHER GUIDANCE FOR CHEMICALS IN GROUNDWATER (µg/L)				
Chemical	Federal Maximum Contaminant Levels	Other Federal Guidance		
		Maximum Contaminant Level goals	Drinking Water Health Advisory for Lifetime Exposure	Secondary Maximum Contaminant Levels
Acrylamide	treatment technique (b)	0 (b)	–	–
Adipates [Di(ethylhexyl)adipate]	500 (P,c)	500 (P,c)	–	–
Alachlor	2 (b)	0 (b)	–	–
Aldicarb	3 (P,i)	1 (P,i)	10 (e)	–
Aldicarb sulfoxide	3 (P,i)	1 (P,i)	10-42 (e,o)	–
Aldicarb sulfone	3 (P,i)	2 (P,i)	10 (e)	–
Aldicarb (parent, sulfoxide, sulfone)	–	1 (P,i)	–	–
Aluminum	–	–	–	50-200 (b)
Ametryn	–	–	60 (d)	–
Ammonium sulfamate	–	–	2,000 (d)	–
Antimony	10; 5 (P,c,l)	3 (P,c)	–	–
Arsenic	50 (a)	50 (P,h)	–	–
Asbestos (fibers/liter >10 um)	7,000,000 (b)	7,000,000 (b)	–	–
Atrazine	3 (b)	3 (b)	3 (d)	–
Barium	1,000 (a) 2,000 (P,i)	2,000 (P,i)	1,500 (e)	–
Baygon	–	–	3 (d)	–
Bentazon	–	–	20 (d)	–
Benzene	5 (a)	0 (a)	–	–
Beryllium	1 (P,c)	0 (P,c)	–	–
Gamma-BHC (Lindane)	0.2 (b)	0.2 (b)	2 (e)	–
Bromacil	–	–	90 (d)	–
Bromochloromethane	100 (a,j)	–	90 (p)	–
Bromoform	100 (a,j)	–	–	–
Bromomethane	–	–	10 (q)	–
2-Butanone (MEK)	–	–	170 (e)	–
Butylate	–	–	700 (d)	–
Cadmium	5 (b)	5 (b)	5 (e)	–
Carbaryl	–	–	700 (d)	–
Carbofuran	40 (b)	40 (b)	36 (e)	–
Carbon tetrachloride	5 (a)	0 (a)	–	–

APPENDIX R (Cont'd)

APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS) AND OTHER GUIDANCE FOR CHEMICALS IN GROUNDWATER (µg/L)				
Chemical	Federal Maximum Contaminant Levels	Other Federal Guidance		
		Maximum Contaminant Level goals	Drinking Water Health Advisory for Lifetime Exposure	Secondary Maximum Contaminant Levels
Carboxin	–	–	700 (d)	–
Carcinogenic PAHs [benzo(a)pyrene]	0.2 (P,c)	0 (P,c)	–	–
Chloramben	–	–	100 (d)	–
Chlordane	2 (b)	0 (b)	–	–
Chloride	–	–	–	250,000 (f)
Chlorobenzene	100 (b)	100 (b)	300 (e)	–
Chloroform	100 (a,j)	–	–	–
bis(2-Chloroisopropyl)ether	–	–	300 (r)	–
Chloromethane	–	–	3 (s)	–
o-Chlorotoluene	–	–	100 (t)	–
p-Chlorotoluene	–	–	100 (u)	–
Chromium	100 (b)	100 (b)	120 (e)	–
Copper	1,300 (P,k)	1,300 (P,k)	–	1,000 (f)
Cyanide	200 (P,c)	200 (P,c)	154 (e)	–
Cyanazine	–	–	10 (d)	–
Dacthal (DCPA)	–	–	4,000 (d)	–
Dalapon	200 (P,c)	200 (P,c)	200 (d)	–
Diazinon	–	–	0.6 (d)	–
1,2-Dibromochloro-3-propane (DBCP)	0.2 (b)	0 (b)	–	–
Dicamba	–	–	200 (d)	–
1,2-Dichlorobenzene	600 (b)	600 (b)	620 (e)	10 (P,g)
1,3-Dichlorobenzene	–	–	620 (e)	–
1,4-Dichlorobenzene	75 (a)	75 (a)	75 (e)	5 (P,g)
Dichlorodifluoromethane	–	–	1,000 (v)	–
1,2-Dichloroethane	5 (a)	0 (a)	–	–
1,1-Dichloroethene	7 (a)	7 (a)	7 (e)	–
cis-1,2-Dichloroethene	70 (b)	70 (b)	70 (e)	–
trans-1,2-Dichloroethene	100 (b)	100 (b)	70 (e)	–
2,4-Dichlorophenoxyacetic acid (2,4-D)	70 (b)	70 (b)	70 (e)	–
1,2-Dichloropropane	5 (b)	0 (b)	–	–
Diethylhexylphthalate	4 (P,c)	0 (P,c)	–	–

APPENDIX R (Cont'd)

APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS) AND OTHER GUIDANCE FOR CHEMICALS IN GROUNDWATER (µg/L)				
Chemical	Federal Maximum Contaminant Levels	Other Federal Guidance		
		Maximum Contaminant Level goals	Drinking Water Health Advisory for Lifetime Exposure	Secondary Maximum Contaminant Levels
Diguat	20 (P,c)	20 (P,c)	–	–
Diisopropyl methylphosphonate (DIMP)	–	–	600 (w)	–
Dimethrin	–	–	2,000 (d,x)	–
Dinoseb	7 (P,c)	7 (P,c)	7 (d)	–
Diphenamid	–	–	200 (d)	–
Disulfoton	–	–	0.3 (d)	–
Diuron	–	–	10 (d)	–
Endothall	100 (P,c)	100 (P,c)	100 (d)	–
Endrin	0.2 (a) 2 (P,c)	2 (P,c)	0.32 (e)	–
Epichlorohydrin	treatment technique (b)	0 (b)	–	–
Ethylbenzene	700 (b)	700 (b)	680 (e)	30 (P,g)
Ethylene dibromide (EDB)	0.05 (b)	0 (b)	–	–
Ethylene glycol	–	–	7,000 (e)	–
Fenamiphos	–	–	2 (d)	–
Fluometuron	–	–	90 (d)	–
Fluoride	4,000 (a)	4,000 (a)	–	2,000 (f)
Fonofos	–	–	10 (d)	–
Glyphosate	700 (P,c)	700 (P,c)	800 (d)	–
Heptachlor	0.4 (b)	0 (b)	–	–
Heptachlor epoxide	0.2 (b)	0 (b)	–	–
Hexachlorobenzene	1 (P,c)	0 (P,c)	–	–
Hexachlorobutadiene	–	–	1 (y)	–
Hexachlorocyclopentadiene (HEX)	50 (P,c)	50 (P,c)	–	8 (P,c)
Hexazinone	–	–	200 (d)	–
HMX	–	–	400 (z)	–
Iron	–	–	–	300 (f)
Lead	50 (a) 5 (P,k)	0 (P,k)	–	–
MCPA	–	–	4 (d)	–
Maleic hydrazide	–	–	4,000 (d)	–

APPENDIX R (Cont'd)

APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS) AND OTHER GUIDANCE FOR CHEMICALS IN GROUNDWATER (µg/L)				
Chemical	Federal Maximum Contaminant Levels	Other Federal Guidance		
		Maximum Contaminant Level goals	Drinking Water Health Advisory for Lifetime Exposure	Secondary Maximum Contaminant Levels
Manganese	–	–	–	50 (f)
Mercury	2 (b)	2 (b)	1.1 (e)	–
Methomyl	–	–	200 (d)	–
Methoxychlor	40 (b)	40 (b)	340 (e)	–
Methyl parathion	–	–	2 (d)	–
Methylene chloride (dichloromethane)	5 (P,c)	0 (P,c)	–	–
Metolachlor	–	–	100 (d)	–
Metribuzin	–	–	200 (d)	–
Naphthalene	–	–	20 (aa)	–
Nickel	100 (P,c)	100 (P,c)	150 (e)	–
Nitrate (as N)	10,000 (b)	10,000 (b)	10,000 (e)	–
Nitrite (as N)	1,000 (b)	1,000 (b)	1,000 (e)	–
Nitrate + Nitrite (as N)	10,000 (b)	10,000 (b)	–	–
Oxamyl [Vydate]	200 (P,c)	200 (P,c)	–	–
Carcinogenic PAHs	0.2 (P,c)	0 (P,c)	–	–
Paraquat	500 (P,c)	500 (P,c)	30 (d)	–
Pentachlorophenol	1 (P,i)	0 (P,i)	220 (e)	30 (P,g)
Picloram	500 (P,c)	500 (P,c)	500 (d)	–
Polychlorinated Biphenyls (PCBs)	0.5 (b)	0 (b)	–	–
Prometon	–	–	100 (d)	–
Pronamid	–	–	50 (d)	–
Propachlor	–	–	90 (d)	–
Propazine	–	–	10 (d)	–
Propham	–	–	100 (d)	–
RDX	–	–	2 (ab)	–
Radionuclides (pCi/liter)				
- Radium 226 and 228	5 (a)	–	–	–
- Gross alpha activity (al)	15 (a)	–	–	–
- Gross beta activity	4 mrem/yr (a)	–	–	–
- Strontium-90	8 (a)	–	–	–
- Tritium	20,000 (a)	–	–	–

APPENDIX R (Cont'd)

APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS) AND OTHER GUIDANCE FOR CHEMICALS IN GROUNDWATER (µg/L)				
Chemical	Federal Maximum Contaminant Levels	Other Federal Guidance		
		Maximum Contaminant Level goals	Drinking Water Health Advisory for Lifetime Exposure	Secondary Maximum Contaminant Levels
Selenium	50 (b)	50 (b)	–	–
Silver	50 (a)	–	–	100 (b)
Simazine	1 (P,c)	1 (P,c)	4 (d)	–
Styrene	100 (b)	100 (b)	140 (e)	10 (P,g)
Sulfate	400,000; 500,000 (P,c,m)	400,000; 500,000 (P,c,m)	–	250,000 (f)
2,3,7,8-TCDD [Dioxin]	0.00005 (P,c)	0 (P,c)	–	–
Tebuthiuron	–	–	500 (d)	–
Terbacil	–	–	90 (d)	–
Terbufos	–	–	0.9 (d)	–
1,1,1,2-Tetrachloroethane	–	–	70 (ac)	–
Tetrachloroethene	5 (b)	0 (b)	10 (e,ad)	–
Thallium	2; 1 (P,c,n)	0.5 (P,c)	–	–
Toluene	1,000 (b)	1,000 (b)	2,420 (e)	40 (P,g)
Toxaphene	3 (b)	0 (b)	–	–
1,2,4-Trichlorobenzene	9 (P,c)	9 (P,c)	9 (ae)	–
1,3,5-Trichlorobenzene	–	–	40 (af)	–
2,4,5-Trichlorophenoxyacetic acid (2,4,5-T)	–	–	70 (d)	–
2,4,5-Trichlorophenoxy-propionic acid (2,4,5-TP)	50 (b)	50 (b)	52 (e)	–
1,1,1-Trichloroethane	200 (a)	200 (a)	200 (e)	–
1,1,2-Trichloroethane	5 (P,c)	3 (P,c)	3 (ag)	–
Trichloroethene	5 (a)	0 (a)	–	–
Trichlorofluoromethane	–	–	2,000 (ah)	–
1,2,3-Trichloropropane	–	–	40 (ai)	–
Trifluralin	–	–	2 (d)	–
Trinitroglycerol (TNG)	–	–	5 (aj)	–
2,4,6-Trinitrotoluene(TNT)	–	–	2 (ak)	–
Vinyl chloride	2 (a)	0 (a)	–	–
Xylenes (total)	10,000 (b)	10,000 (b)	400 (e)	20 (P,g)
Zinc	–	–	–	5,000 (f)

APPENDIX R (Cont'd)

. – = Standard not developed for this chemical.
(P) = Proposed.

(a) 40 CFR, Part 141-National Primary Drinking Water Regulations. 559-563, 620-621.

(b) Environmental Protection Agency (EPA). 1991. National Primary Drinking Water Regulations; Final Rule. Federal Register. Vol. 56, No. 20, Wednesday, January 30, 1991. 3526-3597.

(c) Environmental Protection Agency (EPA). 1990. National Primary and Secondary Drinking Water Regulations; Synthetic Organic Chemicals and Inorganic Chemicals. Proposed Rule. Federal Register. Vol. 53, No. 143, Wed. July 25, 1990.

(d) Environmental Protection Agency (EPA). 1988. Health Advisories for 50 pesticides. Office of Drinking Water. Washington, D.C. August, 1988.

(e) Environmental Protection Agency (EPA). 1987. Health Advisories. Office of Drinking Water. Washington, D.C. March 31, 1987.

(f) 40 CFR, Part 143-National Secondary Drinking Water Regulations. 674.

(g) Environmental Protection Agency (EPA). 1989. National Primary and Secondary Drinking Water Regulations; Proposed Rule. Federal Register. Vol. 54, No. 97, Monday, May 22, 1989. 22062-22160.

(h) Environmental Protection Agency (EPA). 1985. National Primary Drinking Regulations. Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms. Proposed Rule. Federal Register, Vol. 50, No. 219, Wednesday, November 13, 1985. 46935-47025.

(i) Environmental Protection Agency (EPA). 1991. National Primary Drinking Water Regulations - Monitoring for Synthetic Organic Chemicals; MCLGs and MCLs for Aldicarb, Aldicarb Sulfoxide, Aldicarb Sulfone, Pentachlorophenol, and Barium; Proposed Rule. Federal Register. Vol. 56, No. 20, Wednesday, January 30, 1991. 3600-3614.

(j) The value of 100 ug/liter is for total trihalomethanes (i.e., the sum of chloroform, bromochloromethane, and bromoform).

(k) Environmental Protection Agency (EPA). 1988. Drinking Water Regulations; Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper; Proposed Rule. Federal Register. Vol. 53, No. 160, 31515-31578, Thursday, August 18, 1988.

(l) EPA proposes MCLs of 10 ug/l and 5 ug/l for antimony based on proposed practical quantitation levels (PQLs).

(m) EPA proposes MCLs and MCLGs for sulfate of 400,000 ug/l based on Science Advisory Board and WHO guidance and 500,000 ug/l based on studies by Peterson (1951) and Moore (1952) and Canadian guidelines.

(n) EPA proposes MCLs of 2 ug/l and 1 ug/l for thallium based on proposed practical quantitation levels(PQLs)(o) The HA value for the sulfone ranges from 10 to 42 ug/l depending on the presence or absence of other aldicarb/aldicarb sulfoxide residues; only if the sulfone metabolite is present alone as a contaminant, the HA value of 42 ug/l may be used.

(p) Environmental Protection Agency (EPA). 1989. Health Advisory for Bromochloromethane. Office of Drinking Water. Washington, D.C. October, 1989.

(q) Environmental Protection Agency (EPA). 1989. Health Advisory of Bromomethane. Office of Drinking Water. Washington, D.C. September, 1989.

(r) Environmental Protection Agency (EPA). 1989. Health Advisory for bis(2-Chloroisopropyl)ether. Office of Drinking Water. Washington, D.C. September, 1989.

(s) Environmental Protection Agency (EPA). 1989. Health Advisory for Chloromethane. Office of Drinking Water. Washington, D.C. October, 1989.

(t) Environmental Protection Agency (EPA). 1989. Health Advisory for o-Chlorotoluene. Office of Drinking Water. Washington, D.C. September, 1989.

(u) Environmental Protection Agency (EPA). 1989. Health Advisory for p-Chlorotoluene. Office of Drinking Water. Washington, D.C. June, 1989.

(v) Environmental Protection Agency (EPA). 1989. Health Advisory for Dichlorodiflouromethane. Office of Drinking Water. Washington, D.C. September, 1989.

(w) Environmental Protection Agency (EPA). 1989. Health Advisory on Diisopropyl Methylphosphonate (DIMP). Office of Drinking Water. Washington, D.C. January, 1989.

(x) The Lifetime HA of 2,000 ug/l apparently exceeds the water solubility of dimethrin (insoluble).

(y) Environmental Protection Agency (EPA). 1989. Health Advisory for Hexachlorobutadiene. Office of Drinking Water. Washington, D.C. September, 1989.

(z) Environmental Protection Agency (EPA). 1988. Health Advisory for Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). Office of Drinking Water. Washington, D.C. November, 1988.

(aa) Environmental Protection Agency (EPA). 1990. Health Advisory for Naphthalene. Office of Drinking Water. Washington, D.C. March, 1990.

(ab) Environmental Protection Agency (EPA). 1988. Health Advisory for Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX). Office of Drinking Water. Washington, D.C. November, 1988.

(ac) Environmental Protection Agency (EPA). 1989. Health Advisory for 1,1,1,2-Tetrachloroethane. Office of Drinking Water. Washington, D.C. September, 1989.

(ad) Lifetime health advisory was based on the assumption that tetrachloroethene was a Group C carcinogen. Currently, EPA classifies tetrachloroethene as Group B2 - Possible Human Carcinogen.

(ae) Environmental Protection Agency (EPA). 1989. Health Advisory for 1,2,4-Trichlorobenzene. Office of Drinking Water. Washington, D.C. September, 1989.

(af) Environmental Protection Agency (EPA). 1989. Health Advisory for 1,3,5-Trichlorobenzene. Office of Drinking Water. Washington, D.C. September, 1989.

(ag) Environmental Protection Agency (EPA). 1989. Health Advisory for 1,1,2-Trichloroethane. Office of Drinking Water. Washington, D.C. September, 1989.

(ah) Environmental Protection Agency (EPA). 1989. Health Advisory for Trichlorofluoromethane. Office of Drinking Water. Washington, D.C. September, 1989.

(ai) Environmental Protection Agency (EPA). 1989. Health Advisory for 1,2,3-Trichloropropane. Office of Drinking Water. Washington, D.C. September, 1989.

(aj) Environmental Protection Agency (EPA). 1987. Health Advisory for Trinitroglycerol. Office of Drinking Water, Washington, D.C. September, 1987.

(ak) Environmental Protection Agency (EPA). 1989. Health Advisory on 2,4,6-Trinitrotoluene. Office of Drinking Water. Washington, D.C. January, 1989.

(al) Gross alpha particle activity includes radium-226 but excludes radon and uranium.

APPENDIX S

SCREENING LEVEL EVALUATION OF HUMAN EXPOSURES AND RISKS VIA INGESTION OF CROPS AND BEEF FROM MAAP

TABLE OF CONTENTS

APPENDIX S

SCREENING-LEVEL EVALUATION OF HUMAN EXPOSURES AND RISKS ASSOCIATED WITH INGESTION OF CROPS AND BEEF FROM MAAP

This appendix presents a screening-level evaluation of potential human exposures and risks associated with the ingestion of crops and beef raised near the Open Burning Ground (OBG) at MAAP. The OBG is a potential source of contaminated dust emissions at MAAP, and dust released from this area could be transported to areas of MAAP where crops are grown or cattle graze. The exposure and risk assessment methodology used in this appendix follows that outlined in detail in Section 9 of the main report. The remainder of this appendix is organized into five principle sections:

- Section S.1 Exposure Point Concentrations in Crops and Beef
- Section S.2 Human Health Exposure Assessment
- Section S.3 Human Health Toxicity Assessment
- Section S.4 Human Health Risk Assessment
- Section S.5 Summary, Uncertainties, and Conclusions

S.1 EXPOSURE POINT CONCENTRATIONS IN CROPS AND BEEF

This section presents the methodology and equations used to estimate chemical concentrations in locally produced crops and beef for chemicals of potential concern in surface soil at the OBG. The concentrations in these media are dependent on the soil concentrations at the OBG, deposition rates of chemicals transported from the OBG due to wind erosion, and soil and crop concentrations in the crop and pasture areas where chemicals may be deposited.

S.1.1 OBG SOIL CONCENTRATIONS AND CHEMICAL-SPECIFIC DEPOSITION RATES

Concentrations of chemicals were estimated for both human and livestock crops, as well as for pasture grass. Although most agricultural crops currently grown at MAAP are dedicated to livestock feed, it also is possible that crops for human consumption could be grown. Information received from MAAP outlining pasture and crop areas at MAAP (Martin Marietta 1986), was used to determine the areas where crops are grown and where cattle graze. For the purposes of this assessment, it was assumed that particulate matter from the OBG is deposited on the crops and soil in both pasture land and in areas where crops are grown.

The Fugitive Dust Model (FDM), described in Appendix O, was used to estimate ambient air concentrations and deposition rates for fugitive dusts originating from the OBG. Deposition rates were predicted for those areas estimated to receive maximum deposition of particulates. These determinations were made based on consideration of distance from the OBG as well as location relative to predominant wind direction. For crops, deposition rates to 21 receptors located in a line approximately 2 kilometers north of the OBG area were averaged to estimate an average deposition rate to crops. This location was determined by preliminary modeling to be the closest area where crops are grown that the greatest deposition would occur. For pasture land, where cattle graze, deposition rates to 25 receptors located in a line approximately 2.8 kilometers to the west of the OBG were averaged to estimate an average deposition rate to pasture land. Once again, this location was determined by preliminary modeling to be the closest area of pasture land where the greatest deposition would occur.

All major input parameters, as well as the predicted unit air concentrations, used in the model were summarized in Table O-6, in Appendix O. The concentrations were then multiplied by the mass fraction (in g/g) of the concentrations of the chemicals of potential concern in the soil to estimate the chemical-specific concentrations in air where crops and pasture grass are grown. In the same manner, the average deposition rate was multiplied by the mass fraction (in g/g) of chemicals of potential concern in soil to determine the chemical-specific deposition rates to crops and pasture land. These chemical-specific deposition rates are summarized in Table S-1.

It should be noted that the dust transport model does not include two volatile chemicals (acetone and trichlorofluoromethane) which were detected in surface soils at the OBG. These chemicals were detected in a single sample at concentrations of 0.5 mg/kg and 0.02 mg/kg, respectively, and were excluded from the dust transport model because they would preferentially partition in air and would be less likely to remain adhered to particulate matter. Since these two volatile chemicals would not be deposited onto crops, due to their volatile characteristics, they were not included in the models.

S.1.2 CHEMICAL CONCENTRATIONS IN CROP AND PASTURE AREA SOILS

Soil concentrations in the crop and pasture areas were estimated using the area-specific deposition rates described above. The model used to estimate soil concentrations assumes that all materials deposited on land are adsorbed to particulate matter, would be retained in the soil, and would be mixed uniformly into a layer of soil of a specified thickness (conservatively assumed to be 0.15 meters, due to tilling of the soil). All the deposited chemicals were assumed to have accumulated since 1942 (a duration of approximately 50 years) when MAAP was established, neglecting all mechanisms of loss, including photodegradation, biodegradation, volatilization, runoff, resuspension of particulate matter, removal of soil, and mixing to greater depths. This approach is conservative because some or all of these mechanisms are likely to affect actual chemical concentrations in soil.

The following formula was used to calculate chemical concentrations in soil in the crop and pasture areas:

$$C_s = \frac{(DR)\ (AT)\ (X)}{(SD)\ (BD)}$$

where

C_s = concentration of chemical in soil (mg/kg)

DR = chemical-specific deposition rate (g/m^2-yr)

AT = accumulation time (50 yr)

X = conversion factor (1,000 mg/g)

SD = soil depth of mixing (0.15 m)

BD = soil bulk density (1.33×10^3 kg/m^3)

TABLE S-1

ESTIMATED DEPOSITION RATES TO CROPS AND PASTURE LAND
FROM WIND EROSION OF SURFACE SOILS IN THE OBG AREA

Chemical (a)	RME Concentration in Surface Soil (g/g)	Chemical-Specific Deposition Rate (ug/m2-sec) (b)	
		Crops	Pasture Land
Explosives (c):			
HMX (HMX)	3.40E-04	2.38E-06	6.80E-07
Nitrobenzene (NB)	4.30E-06	3.01E-08	8.60E-09
RDX (RDX)	3.30E-03	2.31E-05	6.60E-06
1,3,5-TNB (135TNB)	2.30E-06	1.61E-08	4.60E-09
2,4,6-TNT (246TNT)	4.10E-03	2.87E-05	8.20E-06
Inorganics (d):			
Arsenic (AS)	9.80E-06	4.41E-07	1.08E-07
Chromium (CR)	3.50E-05	1.57E-06	3.85E-07
Lead (PB)	9.30E-05	4.18E-06	1.02E-06
Mercury (HG)	1.40E-06	6.30E-08	1.54E-08
Silver (AG)	1.00E-07	4.50E-09	1.10E-09
Zinc (ZN)	9.48E-05	4.27E-06	1.04E-06

(a) USATHAMA chemical codes are listed in parentheses.
(b) Deposition rates are calculated by multiplying the chemical concentration in soil (in g/g) by the average deposition rate computed by using the Fugitive Dust Model (FDM). The average deposition rate for the nearest crops is 0.045 ug/m2-sec for inorganics and 0.007 ug/m2-sec for explosives. The average deposition rate for the nearest pasture is 0.011 ug/m2-sec for inorganics and 0.002 ug/m2-sec for explosives. The locations of the nearest crops and pasture are discussed in the text.
(c) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, and OBGB-4.
(d) Data used in estimating RME concentrations incorporate sampling locations OBGA-3, OBGA-4, OBGA-6, OBGB-4, OBGB-5, OBGC-4, OBGC-5, OBGD-3, and OBGD-4.

S.1.3 CHEMICAL CONCENTRATIONS IN CROPS AND PASTURE GRASS

Exposures to humans via ingestion of locally grown produce were evaluated using leafy vegetables as a surrogate for all produce. Leafy crops (e.g., cabbage) were selected because they are known to be grown in areas surrounding MAAP and could potentially be grown in the crop area near the OBG (personal communication, Larry Kimmery, May 30, 1991). In addition, since leafy vegetables have larger surface areas, and since deposition of particulate matter onto the crops is a critical factor when assessing chemical concentrations in crops, assessing leafy vegetables as a surrogate for human ingested crops grown in the MAAP area is a conservative selection. The leafy crop category was evaluated, rather than separately evaluating individual produce crops, because detailed information on each specific crop (e.g., the ability to take chemicals up from soil into leaves) is not available in published sources.

In addition to evaluating crops intended for human ingestion, chemical concentrations in pasture grass and crops used as livestock feed also were evaluated. It was assumed that the diet of beef cattle in the area consists of pasture grass which is supplemented by other feed (assumed to be corn silage) (personal communication, Larry Kimmery, May 30, 1991). For the purposes of this assessment, it was conservatively assumed that the cattle graze entirely in the modeled pasture area, and that all of the corn silage they receive is grown in the modeled crop area.

Chemicals transported in dusts can concentrate in plants by two routes. They can be deposited directly on above-ground plant parts and they also can be deposited on the soil, taken up through the root and translocated to other parts of the plant. For leafy crops, corn silage and pasture grass, it is assumed that both deposition and translocation of chemicals may contribute to concentrations in the edible portion of the plant. The area-specific deposition rates and soil concentrations derived for the crop and pasture areas were used in the models described below to estimate inputs from deposition and uptake.

Chemical concentrations in the crops and pasture grass were calculated by the following general formula:

$$C_p = C_{dep} + C_{trans}$$

where

C_p = concentration of chemical in the plant (mg/kg)

C_{dep} = concentration from direct deposition of chemicals onto the plant (mg/kg)

C_{trans} = concentration from translocation of chemicals through the root (mg/kg)

S.1.3.1 Chemical Concentrations Due to Direct Deposition

The contribution due to deposition of chemicals onto plants is calculated based on the methodology of Moghissi et al. (1980):

$$C_{dep} = \frac{(DR)\ (IF)\ (X)\ (DY)}{(k_t)\ (Y)} (1 - \exp[-k_t(T)])$$

where

C_{dep} = concentration from direct deposition of chemicals onto the plant (mg/kg)

DR = chemical-specific deposition rate (g/m^2-year)

IF = interception fraction (unitless)

X = conversion factor (1,000 mg/g)

DY = conversion factor (yr/365 day)

k_t = weathering rate constant (day^{-1})

Y = yield (kg/m^2)

T = growth period (i.e., exposure period of crop) (days)

Interception fractions describe the fraction of deposited particles filtered out of the air by a particular plant, over a unit area. Interception fractions are primarily determined by plant surface area, but are also influenced by such factors as surface shape, folding, and texture. Baes et al. (1984) developed equations for estimating interception fractions for crops based on the observed deposition of radionuclides on plants. The Baes et al. (1984) equations used in this assessment are presented below (an equation developed for hay was used for pasture grass):

$$IF_l = 1 - \exp(-0.0846 Y_l)$$

$$IF_h = 1 - \exp(-2.88 Y_h)$$

$$IF_{cs} = 1 - \exp(-0.769 Y_{cs})$$

where

IF_l = interception fraction for leafy crops (unitless)

IF_h = interception fraction for hay (unitless)

IF_{cs} = interception fraction for corn silage (unitless)

Y_l = yield of leafy crops (kg/m^2)

Y_h = yield of hay per cutting (kg/m^2, dry)

Y_{cs} = yield of corn silage per cutting (kg/m^2, dry)

The yields obtained for leafy crops, pasture grass (hay) and corn silage were 3.5 kg/m^2, 0.4 kg/m^2, and 0.75 kg/m^2, respectively (Baes et al. 1984).

The growth period is the length of time the crop or grass may be in the field and exposed to depositing particles and chemicals present in soil. For both leafy crops as well as corn silage, the growth period was assumed to be approximately 120 days (based on a growing season from mid-May through mid-September), while for pasture grass, the growth period was assumed to be 80 days (assuming the turnover for the grazed pasture grass is approximately four times per year).

The first-order "weathering" rate constant, which pertains to the loss of particulates from plant surfaces, is derived from a half-life estimate of approximately 14 days (k_t = 0.693/14 = 0.0495 day^{-1}) based on experimental observations for particulate radionuclides on plant surfaces (Moghissi et al. 1980). This rate constant does not, however, take into account the loss of deposited chemicals due to degradation or volatilization, and may therefore be underestimated for certain chemicals.

S.1.3.2 Chemical Concentrations Due to Translocation

Chemical concentrations due to translocation can be calculated in two different ways, depending upon the type of data available that characterize the extent of chemical uptake. One set of uptake factors describes the chemical concentration in plants in mg/kg plant per g/m^2 deposited on the soil. The second set of uptake factors describes the chemical concentration in plants in mg/kg plant per mg/kg in soil. In other words, the first set predicts plant concentrations based on deposition rates, while the second predicts plant concentrations based on soil concentration. Often only one type of uptake factor is available for a given chemical and crop.

When using the first type of uptake factor, concentrations from translocation of chemicals through the root can be calculated using the following formula:

$$C_{trans} = (DR)\ (AT)\ (UF_1)$$

where

C_{trans} = concentration from translocation of chemicals through the root (mg/kg)

DR = chemical-specific deposition rate (g/m^2-yr)

AT = accumulation time (50 years)

UF_1 = uptake factor (mg/kg plant divided by g/m^2 chemical deposited on soil)

Using the second type of uptake factor based on soil concentration, concentrations in plants from translocation can be calculated by the equation:

$$C_{trans} = (C_s)\ (UF_2)$$

where

C_{trans} = concentration from translocation of chemicals through the root (mg/kg)

DR = chemical-specific deposition rate (g/m^2-yr)

C_s = concentration of chemical in soil (mg/kg)

UF_2 = uptake factor (mg/kg plant divided by mg/kg soil)

Chemical concentrations in soil in crop and pasture grass areas are calculated assuming a 0.15 m mixing depth, since tilling may be done to this depth. As noted earlier, mechanisms of chemical loss were not considered.

The amount of chemical uptake from soil by the plant, as expressed by the uptake factor, varies with each chemical. In addition, it should be noted that there is a considerable amount of uncertainty regarding the extent of uptake of organic and inorganic chemicals from soil into plants. The uptake factors used in this assessment are described below.

Plant Uptake Factors For Inorganic Chemicals. The uptake factors used for inorganics in this assessment are presented in Table S-2 for leafy crops, corn silage and pasture grass. The average values from crop-specific sludge studies (EPA 1985) are used where available (i.e., UF_1, based on deposition rate), otherwise generic plant uptake values presented by Baes et al. (1984) are used (i.e., UF_2, based on soil concentration). The sludge studies and the work performed by Baes et al. are discussed below.

The uptake of numerous inorganic chemicals has been studied as part of research efforts on contaminants in sludge (EPA 1985). A major assumption in using data from sludge studies is that the chemical in particulate matter would be taken up by plants to the same extent as the chemical applied in sewage sludge. Because humic substances are present in sludge, the metals are likely to be more mobile than when present in a particulate matter matrix and thus sludge-based uptake factors may overestimate concentrations due to translocation from deposited particulate matter. This may result in a slight overestimation of exposures. In these sludge studies, uptake factors are provided in units of mg of chemical/kg dry weight tissue per kg/hectare chemical applied to the soil (i.e., the first type of uptake factor discussed above, UF_1). These uptake factors were modified to predict fresh weight concentrations in the produce by multiplying by the dry/wet fraction of the produce (Watt and Merrill 1963).

Fresh weight uptake factors based on the sludge studies were available for several of the chemicals of concern (e.g., EPA 1985, John 1972, Furr et al. 1976 and 1981, Chaney et al. 1982, MacLean 1974, and Spittler and Feder 1979). Results from the studies were averaged across individual plant types (i.e., specific leafy crops, various types of pasture grass, and specific corn plant components) in order to derive uptake factors for leafy crops, corn silage and pasture grass in general. These values are often uncertain because of small sample sizes, the use of nonsludge applications (e.g., $HgCl_2$ and mercury fungicide), and the use of data from produce grown in pots rather than in the field.

Uptake factors for inorganics for which no data were available in the sludge studies were based on chemical concentrations from soil data. Generic soil:plant partition coefficients have been developed for inorganic chemicals by the Oak Ridge National Laboratory (Baes et al. 1984). These partition coefficients are presented as dry weight crop concentrations over dry weight soil concentrations. For those chemicals for which uptake factors could not be estimated based on the EPA (1985) sludge studies, the uptake factors provided in Baes et al. (1984) were used (after adjusting to wet weight). It should be noted that

TABLE S-2

PLANT UPTAKE FACTORS FOR THE CHEMICALS OF CONCERN
IN SURFACE SOIL AT THE OBG (a)

Chemical	Leafy Crops		Pasture Grass		Corn Silage	
Organics:(b)						
HMX	1.43E+01	S	1.43E+01	S	1.43E+01	S
Nitrobenzene	6.37E+00	S	6.37E+00	S	6.37E+00	S
RDX	7.19E+00	S	7.19E+00	S	7.19E+00	S
1,3,5-TNB	5.25E+00	S	5.25E+00	S	5.25E+00	S
2,4,6-TNT	2.73E+00	S	2.73E+00	S	2.73E+00	S
Inorganics: (c)						
Arsenic	5E-03	S	5E-02	S	5E-02	S
Chromium	2E-02	D	2E-05	D	4E-02	D
Lead	7E-03	D	3E-02	D	7E-03	D
Mercury	8E-03	D	1E+00	S	1E+00	S
Silver	5E-02	S	4E-01	S	2E-01	S
Zinc	3E-01	D	2E+00	S	3E-01	D

(a) Uptake factors flagged with a "D" are in units of mg/kg fresh weight per g/m2 applied (e.g., UF1). Those flagged with an "S" are in units of mg/kg fresh weight per mg/kg soil (e.g., UF2).

(b) Source: Uptake factors are based on an average of results from the Topp et al. (1986) and Travis and Arms (1988) equations.

(c) Sources: USEPA (1985a) for uptake factors in units of mg/kg fresh weight per g/m2 applied; Baes et al. (1984) for uptake in units of mg/kg fresh weight per mg/kg soil.

because of uncertainty due to a three-order-of-magnitude difference in sludge uptake factors for arsenic in leafy crops from two available studies, the Baes et al. (1984) uptake factor for arsenic in leafy crops was used.

Plant Uptake Factors for Organic Chemicals. The database for organic chemicals is less reliable than that for inorganics. Experimental results support the observation that translocation may occur and that the extent of translocation differs by plant species and parts. Empirical or experimental values determined from field, microcosm, and laboratory experiments should be used to determine plant uptake whenever possible. In their absence, some equations have been presented for use when estimating uptake of organic chemicals into the edible portion of plants.

The equations for whole plant uptake factors developed by Topp et al. (1986) and Travis and Arms (1988) were conservatively used in this assessment. The semi-empirical regression equation developed by Topp et al. (1986) is as follows:

$$\log(UF_{Topp}) = 5.943 - 2.385 \log(M)$$

or, rearranged:

$$UF_{Topp} = (8.77x10^{5}) \times M^{(-2.385)}$$

where:

UF_{Topp} = uptake factor (mg/kg plant per mg/kg soil)

M = molecular weight (g/mole)

The equation developed by Travis and Arms (1988) is as follows:

$$UF_{T-A} = 10^{(1.588 - 0.578 * Log K_{ow})}$$

where:

UF_{T-A} = uptake factor (mg/kg plant per mg/kg soil, or UF_2)

K_{ow} = octanol:water partition coefficient

Molecular weights and log K_{ow} values are presented in Table S-3. The values produced by the Topp et al. (1986) and Travis and Arms (1988) equations were arithmetically averaged to produce the uptake factors for the crops used in this assessment:

$$UF_2 = \frac{UF_{Topp} + UF_{T-A}}{2}$$

Uptake factors for organic chemicals are also presented in Table S-2.

TABLE S-3

LOG Kow VALUES AND MOLECULAR WEIGHTS OF THE ORGANIC CHEMICALS OF CONCERN IN OBG SURFACE SOILS

Chemical	Log Kow	Molecular Weight
HMX	0.26	296.2
Nitrobenzene	1.85	121.11
RDX	0.87	222.15
1,3,5-TNB	1.18	213.12
2,4,6-TNT	1.84	227.15

Source: Burrows et al. (1989).

S.1.4 CHEMICAL CONCENTRATIONS IN LOCALLY PRODUCED BEEF

As discussed in Section 9, some beef and dairy cattle are raised in the MAAP area, although beef cattle are more prevalent (personal communication, Larry Kimmery, May 30, 1991). To estimate exposures to residents due to ingestion of locally raised beef, chemical concentrations in the environmental media that beef cattle may be exposed to must first be estimated. It was assumed that cattle would be exposed to chemicals via the ingestion of pasture grass and corn silage, as well as via incidental ingestion of soil while grazing in pasture areas. The approach and equations used to estimate chemical concentrations in soil in the pasture area, pasture grass and corn silage were described in previous sections. In this section, the approach and equations used to estimate chemical concentrations in beef are described.

The concentration of a chemical in beef is related to the total mass of chemical ingested per day. To obtain the total amount of ingested chemical per day, the animal's daily intake of corn silage, pasture grass and soil is multiplied by the chemical concentrations in corn silage, pasture grass and soil, respectively. The equation that estimates the concentration of the chemical in beef as a result of the ingestion of several food and non-food sources is shown below, adapted from Moghissi et al. (1980):

$$C_{meat} = (L)\,(F_{meat})\,(Bio)\,\sum_{i=1}^{x} C_i Q_i$$

where

C_{meat} = concentration of chemical in meat (mg chemical/kg meat)

L = fraction of feed crops grown locally (1.0)

F_{meat} = feed-to-meat transfer coefficient (day/kg)

Bio = fraction of the contaminant in a particular media which is bioavailable (dimensionless)

x = total number of food or non-food sources i (pasture grass, soil)

C_i = concentration of chemical in source i (mg chemical/kg source)

Q_i = quantity of source i ingested per head of cattle per day (kg/day)

Beef cattle were assumed to eat 5 kg/day of pasture grass, and 5 kg/day of corn silage. While grazing, cattle also ingest soil, and thus the chemical concentrations estimated earlier for pasture area soil were also used to estimate chemical concentrations in beef. It was assumed that four percent (Fries 1987) of a beef cow's total dry matter intake would be soil.

Not all of the chemical that a beef cow is exposed to will be transferred to meat. An important parameter in the above equation that reflects this fact is the feed-to-meat transfer coefficient, which is used in estimating the chemical concentrations (in mg/kg) in beef at a steady state. Transfer coefficients ideally should be obtained from controlled animal feeding studies that measure the concentration of contaminant in meat or fat as a result of a long term exposure or under steady-state conditions. Based on such studies, a transfer coefficient can be calculated by the following equation (Ng et al. 1979):

$$F_i = \frac{C_i}{(I)}$$

where

F_i = the transfer coefficient from feed to animal meat (day/kg)

C_i = measured chemical concentration in animal meat (ng/kg)

I = reported daily intake of the chemical (ng/day)

However, cattle feeding studies have not been conducted for the organic chemicals being considered in this assessment (i.e., explosives). As a result, transfer coefficients were estimated from regression equations based upon log K_{ow} values. Although regression equations may be inaccurate in estimating transfer coefficients, an equation for beef has been developed (Travis and Arms 1988) which has been found to compare well with feeding studies that are available. This equation is shown below in a modified form to adjust for the fact the authors assumed a 25% fat content in beef, while a 17% fat content in cooked beef was assumed for this assessment:

$$F_{meat} = (10^{-7.6+\log K_{ow}})\,(0.17/0.25)$$

where

F_{meat} = feed-to-meat transfer coefficient based on 17% fat content of cooked beef (days/kg)

K_{ow} = octanol-water partition coefficient for the chemical of concern

Transfer coefficients derived using this equation are presented in Table S-4. Transfer coefficients for inorganics are derived from Baes et al. (1984) except for one value (for mercury) which was obtained from Ng et al. (1982). This mercury value is a feed-to-chicken value which is used because the only other transfer coefficients available for mercury are default values. Transfer coefficients for inorganics are also presented in Table S-4.

Since the transfer coefficients were developed from studies in which chemicals fed to cattle were readily digestible, the intakes of the chemicals by beef cattle via the ingestion of soil, corn silage and pasture grass should be modified by relative bioavailability factors (Bio) to adjust for any matrix effects on digestion or availability. The chemicals in corn silage and pasture grass due to uptake are conceptually in a food matrix and were assumed to be as bioavailable as the chemicals administered in the feeding studies (i.e., Bio = 1). However, the chemicals in corn silage and pasture grass due to deposition, and the chemicals in soil are conceptually bound in a particulate matter matrix and would be less bioavailable. However, for the purposes of this assessment, it was conservatively assumed that the particulate matter matrix would not interfere with bioavailability, and chemicals present in soil or on plants due to deposition would also be as available as chemicals administered in the feeding studies. Since a particulate matter matrix will bind certain chemicals, this assumption will result in an overestimate of chemical concentrations in beef.

TABLE S-4

TRANSFER COEFFICIENTS FOR BEEF

Chemical	Feed-to-Beef Transfer Coefficients (days/kg)
Organics (a)	
HMX	3.11E-08
Nitrobenzene	1.21E-06
RDX	1.27E-07
1,3,5-TNB	2.59E-07
2,4,6-TNT	1.18E-06
Inorganics (b)	
Arsenic	2.0E-03
Chromium	5.5E-03
Lead	3.0E-04
Mercury	2.7E-02 (c)
Silver	3.0E-03
Zinc	1.0E-01

(a) Calculated using a regression equations based on octanol water partition coefficients (Log Kows) (Travis and Arms 1988).
(b) Based on Baes et al. (1984).
(c) Based on Ng et al. (1982). Value is a feed-to-chicken value, conservatively used since the only other value available was a default.

S.2 HUMAN HEALTH EXPOSURE ASSESSMENT

This section presents quantitative exposure estimates (CDIs) for the ingestion of crops and beef by humans. As described in the introduction, chemicals in the OBG surface soils may be transported to areas where crops and pasture grass are grown. Humans may be exposed to these chemicals via ingestion of crops grown in the areas surrounding the OBG, as well as via ingestion of beef that have ingested pasture grass and livestock feed also grown in areas surrounding the OBG.

S.2.1 INGESTION OF LEAFY CROPS

CDIs were calculated for ingestion of leafy crops using the estimated exposure point concentrations derived above as well as the exposure parameters presented in Table S-5 and discussed below.

Leafy crop ingestion exposures were evaluated for persons between the ages of 1 and 30. This age period was conservatively chosen for evaluation because predicted exposures for this age period will be greater than those estimated for persons within other age groups spanning a 30 year period. A time-weighted average body weight of 48 kg (based on data in EPA [1989b]) and a leafy crop ingestion rate of 261 g/day were used as parameters for the exposure scenario. The leafy crop ingestion rate was derived using rates from USDA (1982), which were an ingestion rate of 170 g/day for individuals up to 8 years of age, and an ingestion rate of 294 g/day for those 9 and older. Exposed individuals were assumed to eat leafy crops from impacted crop land for 24 days/year (3 days per week over a harvest period of two months), and obtain 10% of their leafy crops from MAAP. Individuals were assumed to be exposed for 30 years (the upperbound estimate of the time a person is likely to spend in any one residence [EPA 1989a]) and assumed to live for 70 years (EPA 1989a).

Exposures due to leafy crop ingestion were calculated using the above assumptions and the following equation:

$$CDI = \frac{(C_c)\,(IR)\,(Y)\,(EF)\,(ED)\,(FL)\,(Bio)\,(Abs)}{(BW)\,(DY)\,(YL)}$$

where

CDI = chronic daily intake (mg/kg-day)

C_c = exposure point concentration in leafy crops (mg/kg)

IR = ingestion rate of leafy crops (g/day)

Y = conversion factor ($kg/10^3$)

EF = exposure frequency (days/year)

ED = exposure duration (years)

FL = fraction of leafy crops ingested that are locally produced (unitless)

Bio = relative oral bioavailability factor (unitless)

TABLE S-5

PARAMETERS USED TO ESTIMATE
LEAFY CROP INGESTION

Parameter	Value
Exposure Frequency (a)	24 days/year
Exposure Duration (b)	30 years
Ingestion Rate (c)	261 g/day
Body Weight (d)	48 kg
Relative Oral Bioavailability Factor (e)	1.0
Absolute Absorption (Arsenic only) (f)	0.8

(a) Based on residents ingesting leafy crops 3 days a week for an assumed harvest period of 8 weeks.
(b) Based on the upperbound time period that a person is likely to remain at the same residence (EPA 1989a).
(c) Based on ingestion rates provided by USDA (1982): 170 g/day for individuals up to 8 years old and 294 g/day for individuals 9 years of age and older.
(d) Average value for persons between ages of 0-30 (EPA 1989b).
(e) Assumed value for all chemicals.
(f) EPA (1984).

Abs = absorption factor (unitless, for arsenic only)

BW = average body weight over period of exposure (kg)

DY = days in a year (365 days/year)

YL = period over which risk is being estimated (a lifetime [70 years] for potential carcinogens and the period of exposure for noncarcinogens [30 years])

Chemicals in leafy crops were assumed to be 100% bioavailable (i.e., Bio = 1.0). An additional absorption factor was applied when calculating CDIs for arsenic, since arsenic's toxicity criteria are based on an absorbed dose. The absorption of arsenic in the gut was assumed to be 80% (i.e., Abs = 0.8) based on EPA (1984).

To be conservative, no reductions in concentrations were assumed for the prepared crops (leafy produce) due to washing, cutting or chopping. It was assumed that the leafy produce would be eaten raw, since chemical concentrations are likely to be higher on or in raw rather than on or in cooked vegetables. Table S-6 presents the chemical concentrations that were estimated in leafy crops and the resulting CDIs that were derived using the assumed exposure parameters for ingestion of leafy crops.

S.2.2 INGESTION OF BEEF

CDIs were calculated for the ingestion of beef using the exposure point concentrations derived in Section S.1.4 and the beef ingestion exposure parameters presented in Table S-7 and discussed below.

Beef ingestion exposures were also evaluated for persons between the ages of 1 and 30. As discussed for leafy crops, this age period was conservatively chosen for evaluation because exposures for this age period are greater than those estimated for persons 18 and older. Average body weights for the age periods were based on data provided in EPA (1989b), and ingestion rates were derived from USDA (1982). Individuals aged 1-8 were assumed to have an average body weight of 20 kg and an ingestion rate of 92 g/day; individuals aged 9 to 18 were assumed to have an average body weight of 52 kg and an ingestion rate of 152 g/day; individuals 19 years and older were assumed to have an average body weight of 70 kg and an ingestion rate of 166 g/day. All exposed individuals were assumed to eat beef 3 times a week throughout the year (156 days/year), and obtain 44% of their beef from cattle raised at MAAP (EPA 1989b). Individuals were assumed to be exposed for 30 years (the upperbound estimate of the time a person is likely to spend in any one residence [EPA 1989a]) and assumed to live for 70 years (EPA 1989a).

Exposures due to beef ingestion were calculated using the above assumptions and the following equation:

$$CDI = \frac{(C_b)\,(IR)\,(Y)\,(EF)\,(ED)\,(FL)\,(Bio)\,(Abs)}{(BW)\,(DY)\,(YL)}$$

where

CDI = chronic daily intake (mg/kg-day)

C_b = exposure point concentration in beef (mg/kg)

TABLE S-6

ESTIMATES OF CHRONIC INGESTION INTAKES FOR LEAFY CROPS
FOR CHEMICALS OF POTENTIAL CONCERN IN SURFACE SOILS AT THE OBG

CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	LEAFY CROP CONCENTRATION (a) (mg/kg)	CHRONIC LEAFY CROP INGESTION INTAKES (mg/kg-day)
Organic Chemicals		
RDX	1.32E+00	1.98E-05
2,4,6-TNT	6.22E-01	9.37E-06
Inorganic Chemicals		
Arsenic	7.36E-05	8.88E-10

CHEMICALS EXHIBITING NONCARCINOGENIC EFFECTS	LEAFY CROP CONCENTRATION (a) (mg/kg)	CHRONIC LEAFY CROP INGESTION INTAKES (mg/kg-day)
Organic Chemicals		
HMX	2.69E-01	9.44E-06
Nitrobenzene	1.52E-03	5.34E-08
RDX	1.32E+00	4.63E-05
1,3,5-TNB	6.70E-04	2.36E-08
2,4,6-TNT	6.22E-01	2.19E-05
Inorganic Chemicals (b)		
Arsenic	7.36E-05	2.07E-09
Chromium	2.50E-04	8.78E-09
Mercury	8.83E-06	3.10E-10
Silver	2.35E-06	8.27E-11
Zinc	2.56E-03	9.01E-08

(a) The estimated concentrations in leafy crops reflect both direct deposition onto and translocation from soil into the vegetables.
(b) Lead is not evaluated, as toxicity criteria are not available for this chemical.

TABLE S-7

PARAMETERS USED FOR
BEEF INGESTION EXPOSURE ESTIMATES

Parameter	Value
Exposure Frequency (a)	156 days/year
Exposure Duration (b)	30 years
Ingestion Rate (c)	
1-8	92 g/day
9-18	152 g/day
19+	166 g/day
Body Weight (d)	
1-8	20 kg
9-18	52 kg
19+	70 kg
Relative Oral Bioavailability Factor (e)	1.0
Absolute Absorption (Arsenic only) (f)	0.8

(a) Based on eating beef 3 days per week throughout the year.
(b) Based on the upperbound time period that a person is likely to remain at the same residence (EPA 1989a).
(c) Derived from USDA (1982).
(d) Based on EPA (1989b).
(e) Assumed value for all chemicals.
(f) EPA (1984).

IR = ingestion rate of beef (g/day)

Y = conversion factor ($kg/10^3$)

EF = exposure frequency (days/year)

ED = exposure duration (years)

FL = fraction of beef ingested that is locally produced (unitless)

Bio = relative oral bioavailability factor (unitless)

Abs = absorption factor (unitless, for arsenic only)

BW = average body weight over period of exposure (kg)

DY = days in a year (365 days/year)

YL = period over which risk is being estimated (a lifetime [70 years] for potential carcinogens and the period of exposure for noncarcinogens [30 years])

Chemicals in beef were assumed to be 100% bioavailable (i.e., Bio = 1.0). An additional absorption factor was applied when calculating CDIs for arsenic, since arsenic's toxicity criteria are based on an absorbed dose. The absorption of arsenic in the gut was assumed to be 80% based on a dietary study (EPA 1984). To be conservative, no reductions in concentrations were assumed for the preparation of the beef (e.g., cooking). Table S-8 presents the chemical concentrations that were estimated in beef and the resulting CDIs that were derived using the assumed exposure parameters for ingestion of beef.

S.3 HUMAN HEALTH TOXICITY ASSESSMENT

The toxicity of the chemicals of potential concern in leafy crops and beef has been presented in detail in Section 9.3 of the main report. Table S-9 shows the oral critical toxicity values which were derived for the chemicals of potential concern. Toxicity summaries for these chemicals are included in Appendix P.

S.4 HUMAN HEALTH RISK ASSESSMENT

This section presents quantitative risk estimates for the leafy crop and beef ingestion pathways. To calculate risks, the chronic daily intakes estimated for each chemical of potential concern were combined with the health effects criteria (slope factors or RfDs). A more detailed discussion of derivation of risk estimates is presented in Section 9.4 of the main report.

Risks associated with the potential ingestion of leafy crops grown in the crop area near the OBG are shown in Table S-10. The excess lifetime cancer risk estimate associated with the ingestion of leafy crops is $2x10^{-6}$, due primarily to RDX. The hazard index for ingestion of leafy crops is less than 1, thus adverse noncarcinogenic effects are unlikely to occur.

TABLE S-8

ESTIMATES OF CHRONIC BEEF INGESTION INTAKES FOR
CHEMICALS OF POTENTIAL CONCERN FORM SURFACE SOILS OF THE OBG

CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	CONCENTRATION IN BEEF (mg/kg)	CHRONIC BEEF INGESTION INTAKES (mg/kg-day)
Organic Chemicals		
RDX	1.10E-06	2.79E-10
2,4,6-TNT	5.04E-06	1.28E-09
Inorganic Chemicals		
Arsenic	1.05E-05	2.13E-09

CHEMICALS EXHIBITING NONCARCINOGENIC EFFECTS	CONCENTRATION IN BEEF (mg/kg)	CHRONIC BEEF INGESTION INTAKES (mg/kg-day)
Organic Chemicals		
HMX	5.43E-08	3.22E-11
Nitrobenzene	1.21E-08	7.19E-12
RDX	1.10E-06	6.51E-10
1,3,5-TNB	1.15E-09	6.83E-13
2,4,6-TNT	5.04E-06	2.99E-09
Inorganic Chemicals		
Arsenic	1.05E-05	4.97E-09
Chromium	8.43E-05	4.99E-08
Mercury	9.97E-05	5.91E-08
Silver	2.86E-07	1.70E-10
Zinc	1.33E-02	7.85E-06

(a) The concentrations in beef reflect ingestion of pasture grass and corn silage as well as incidental ingestion of soil by a beef cow.
(b) Lead is not evaluated, as toxicity criteria are not available for this chemical.

TABLE S-9

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN AT THE OBG

Chemical	Chronic Reference Dose (mg/kg-day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg-day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals:							
HMX	5.0E-02	1000	Liver	IRIS	--	D	IRIS
Nitrobenzene	5.0E-04	10,000	Kidney/Liver	IRIS	--	D	IRIS
RDX	3.0E-03	100	Prostate	IRIS	1.1E-01	C	IRIS
1,3,5-TNB	5.0E-05	10,000	Spleen	IRIS	--	--	--
2,4,6-TNT	5.0E-04	1,000	Liver	IRIS	3.0E-02	C	IRIS
Inorganic Chemicals:							
Arsenic	1.0E-03	1	Skin	HEAST	2.0E+00 (d)	A	IRIS
Chromium VI and compounds	5.0E-03	500	CNS	IRIS	--	--	IRIS
Lead	--	--	--	IRIS	--	B2	IRIS
Mercury, inorganic	3.0E-04	1,000	Kidney	HEAST	--	D	IRIS
Silver	3.0E-03	2	Skin (Argyria)	IRIS	--	--	--
Zinc	2.0E-01	10	Blood (Anemia)	HEAST	--	--	--

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10-fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- a 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.

'odifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.
target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemical is listed.

(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies;
[C] = Possible human carcinogen based on limited evidence from animal studies in the absence of human studies; and
[D] = Not classified as to human carcinogenicity.

(d) EPA 1988. Special report on ingested inorganic arsenic skin cancer; nutritional essentiality. Risk assessment forum. EPA, Washington, D.C. EPA/625/3-87/013F. July 1988.

NOTE: IRIS = Integrated Risk Information System - March 1, 1991.
HEAST = Health Effects Assessment Summary Tables - First Quarter 1991.
-- = No information available.

TABLE S-10

ESTIMATED RISKS ASSOCIATED WITH INGESTION OF LOCALLY GROWN LEAFY CROPS
FOR CHEMICALS OF POTENTIAL CONCERN FROM SURFACE SOILS AT THE OBG

CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	CHRONIC LEAFY CROP INGESTION INTAKES (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)-1 [Weight-of-Evidence] (a)	INDIVIDUAL EXCESS LIFETIME CANCER RISK
Organic Chemicals			
RDX	1.98E-05	1.10E-01 [C]	2.2E-06
2,4,6-TNT	9.37E-06	3E-02 [C]	2.8E-07
Inorganic Chemicals			
Arsenic	8.88E-10	2E+00 [A]	1.8E-09
Total			2E-06

CHEMICALS EXHIBITING NONCARCINOGENIC EFFECTS	CHRONIC LEAFY CROP INGESTION INTAKES (mg/kg-day)	REFERENCE DOSE (RfD) (mg/kg-day) [Uncertainty Factor]	INTAKE:RfD RATIO
Organic Chemicals			
HMX	9.44E-06	5E-02 [1,000]	1.9E-04
Nitrobenzene	5.34E-08	5E-04 [10,000]	1.1E-04
RDX	4.63E-05	3E-03 [100]	1.5E-02
1,3,5-TNB	2.36E-08	5E-05 [10,000]	4.7E-04
2,4,6-TNT	2.19E-05	5E-04 [1,000]	4.4E-02
Inorganic Chemicals (b)			
Arsenic	2.07E-09	1E-03 [1]	2.1E-06
Chromium	8.78E-09	5E-03 [500](c)	1.8E-06
Mercury	3.10E-10	3E-04 [1,000]	1.0E-06
Silver	8.27E-11	3E-03 [2]	2.8E-08
Zinc	9.01E-08	2E-01 [10]	4.5E-07
Hazard Index (Total) (d)			<1 (6E-02)

(a) USEPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies.
[C] = Limited evidence of carcinogenicity in animals in the absence of data on humans.
(b) Lead is not evaluated, as no toxicity criteria are available for this chemical.
(c) RfD reported is for chromium VI, as all chromium is conservatively assumed to be in the more toxic form of chromium VI.
(d) Hazard index is the sum of the intake:RfD ratios for each chemical.

Risks associated with the ingestion of beef raised near the OBG are shown in Table S-11. The excess lifetime cancer risk estimated for the ingestion of beef is $4x10^{-9}$. The hazard index for the ingestion of beef is well below 1.

S.5 SUMMARY, UNCERTAINTIES, AND CONCLUSIONS

Concentrations of chemicals were estimated for both human and livestock crops, as well as for beef. The screening level model assumed that wind-blown dusts from the OBG are deposited on the crops and soil found on both pasture land used for cattle grazing and on land used for growing livestock feed and leafy crops for human consumption. Although most agricultural crops currently grown at MAAP are dedicated to livestock feed, it also is possible that crops for human consumption could be grown.

A certain amount of uncertainty accompanies the risk estimates derived in this section. Primarily, risks were estimated using very conservative assumptions and therefore are likely to greatly overestimate the actual risks that could occur. Conservative assumptions concerning exposure point concentration models and exposure parameters contributed to the overestimation of risks.

For example, chemical concentrations in soil were likely overestimated as processes such as runoff, resuspension of particulate matter, removal of soil, and mixing of chemicals to greater depths were not considered in the model. Further, not including degradation factors for the organic chemicals greatly overestimates the actual concentrations of the explosive chemicals both in soil and on the plant (i.e., for deposition calculations). When estimating organic chemical concentrations, degradation factors such as photolysis (which can be significant for explosive compounds), biodegradation and volatilization were not taken into account. These assumptions are conservative because some or all of these mechanisms are likely to affect actual chemical concentrations in soil and on the plants.

Further, estimated exposures were probably overestimated because it was assumed that food crops are grown there every year for 30 years. As noted in Section 9 of the main report, however, most (if not all) crops grown at MAAP are dedicated to livestock feed. Thus there is uncertainty with the assumption that leafy crops are grown there at all, or would be during a 30 year period.

There is also a great deal of uncertainty in the predicted cancer risks for Class C carcinogens (possible human carcinogens for which there is limited evidence of carcinogenicity in animals). For ingestion of leafy crops, the risk was driven by RDX and 2,4,6-TNT, both of which are Class C carcinogens. Finally, there is uncertainty with the assumption that chemicals ingested with soil (by cattle) or on plants (by humans or cattle) are as fully available as chemicals administered in toxicity or feeding studies. Chemicals that are deposited onto plants or soil are not 100% bioavailable when ingested, as was assumed in this evaluation. In reality, only a fraction of the chemical is available to be absorbed into the body because of the effects of a soil or particulate matter matrix. Therefore, concentrations of chemicals of concern in cattle feed were likely overestimated.

In light of the conservative nature of this screening analysis, the risks to human receptors via the ingestion of leafy crops grown near the OBG or beef raised at MAAP are likely to be negligible. The risks that were derived indicate that an individual ingesting locally grown leafy crops or beef raised at MAAP are within or lower than the 10^{-6} to 10^{-4} EPA target risk range. The hazard indices for noncarcinogenic effects associated with either pathway are much lower than one, indicating that noncarcinogenic effects are unlikely to occur.

TABLE S-11

ESTIMATED RISKS ASSOCIATED WITH INGESTION OF BEEF
FOR CHEMICALS OF POTENTIAL CONCERN FROM SURFACE SOILS AT THE OBG

CHEMICALS EXHIBITING POTENTIAL CARCINOGENIC EFFECTS	CHRONIC BEEF INGESTION INTAKES (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)-1 [Weight-of-Evidence] (a)	INDIVIDUAL EXCESS LIFETIME CANCER RISK
Organic Chemicals			
RDX	2.79E-10	1.10E-01 [C]	3.1E-11
2,4,6-TNT	1.28E-09	3E-02 [C]	3.8E-11
Inorganic Chemicals			
Arsenic	2.13E-09	2E+00 [A]	4.3E-09
Total			4E-09

CHEMICALS EXHIBITING NONCARCINOGENIC EFFECTS	CHRONIC BEEF INGESTION INTAKES (mg/kg-day)	REFERENCE DOSE (RfD) (mg/kg-day)(a) [Uncertainty Factor]	INTAKE:RfD RATIO
Organic Chemicals			
HMX	3.22E-11	5E-02 [1,000]	6.44E-10
Nitrobenzene	7.19E-12	5E-04 [10,000]	1.44E-08
RDX	6.51E-10	3E-03 [100]	2.17E-07
1,3,5-TNB	6.83E-13	5E-05 [10,000]	1.37E-08
2,4,6-TNT	2.99E-09	5E-04 [1,000]	5.97E-06
Inorganic Chemicals (b)			
Arsenic	4.97E-09	1E-03 [1]	4.97E-06
Chromium	4.99E-08	5E-03 [500](c)	9.99E-06
Mercury	5.91E-08	3E-04 [1,000]	1.97E-04
Silver	1.70E-10	3E-03 [2]	5.65E-08
Zinc	7.85E-06	2E-01 [10]	3.92E-05
Hazard Index (Total)(d)			<1 (3E-04)

(a) USEPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies.
[C] = Possible Human Carcinogen based on limited evidence from animal studies in the absence of human studies.
(b) Lead is not evaluated, as no toxicity criteria are available for this chemical.
(c) RfD reported is for chromium VI, as all chromium is conservatively assumed to be in the more toxic form of chromium VI.
(d) Hazard index is the sum of the intake:RfD ratios for each chemical.

S.6 REFERENCES

BAES, C.F., III, SHARP, R.D., SJOREEN, A.L., and SJORE, R.W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Prepared for the U.S. Department of Energy. ORNL-5786. September 1984.

BURROWS, E.P., ROSENBLATT, D.H., MITCHELL, W.R., and PARMER, D.L. 1989. Technical Report 8901. Organic Explosives and Related Compounds: Environmental and Health Considerations.

CHANEY, R.L., STERRETT, S.B., and MIELKE, H.W. 1984. The potential for heavy-metal exposure from urban gardens and soils. In: Proceedings Symp. on Heavy Metals in Urban Gardens, Dist. Columbia Extension Service, Washington, D.C. Pp. 37-84.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Inorganic Arsenic. Final Report. Office of Health and Environmental Assessment, Washington, D.C. March 1984. EPA-600/8-83-021F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge. Office of Water Regulations and Standards, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989a. Risk Assessment Guidance for Superfund. Vol. I: Human Health Evaluation Manual. Interim Final. Office of Emergency and Remedial Response. Washington, D.C. OSWER Directive 9285.7-01a.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989b. Exposure Factors Handbook. Office of Health and Environmental Assessment. Washington, D.C. EPA/600/8-89/043.

FRIES, G.F. 1987. Assessment of potential residues in foods derived from animals exposed to TCDD-contaminated soil. Pesticide degradation Laboratory. Agricultural Research Service. U.S. Department of Agriculture. Beltsville, Maryland. Chemosphere. 16:8-9.

FURR, A.K., KELLY, W.C., BAEHE, C.A., GUTENMANN, W.H., and LISK, D.H. 1976. Multi-element absorption of crops grown on Ithaca sludge-amended soil. Bull. Env. Contam. Toxicol. 16:756-763. As cited in EPA (1985).

JOHN, M.K. 1972. Mercury uptake from soil by various plant species. Bull. Env. Contam. Toxicol. 8:77. As cited in EPA (1985).

KIMMERY, L. 1991. Gibson County Agricultural Office. May 30, 1991.

MARTIN MARIETTA. 1986. Agricultural Outleasing Map. Drawing Number MA-C-426-6Martin Marietta, Milan Army Ammunition Plant, Milan Tennessee, 11/86.

MacLEAN, A.J. 1974. Mercury in plants and retention of mercury by soils in relation to properties and added sulfur. Am. J. Soil Sci. 54:287. As cited in EPA (1985).

MOGHISSI, A.A., MARLAND, R.E., CONGEL, F.J., and ECKERMAN, K.F. 1980. Chapter 31: Methodology for environmental human exposure and health risk assessment. In: Hague, R. (ed.). Dynamics, Exposure, and Hazard Assessment of Toxic Chemicals. Ann Arbor Science.

NG, Y.C., COLSHER, C.S., and THOMPSON, S.E. 1982. Transfer coefficients for assessing the dose from radionuclides in meat and eggs. Prepared for the U.S. Nuclear Regulatory Commission. NTIS NVREG/CR-2976.

SPITTLER, T.M. and FEDER, W.A. 1979. A study of soil contamination and plant lead uptake in Boston urban gardens. Commun. Soil Sci. Plant Anal. 10:1195. As cited in EPA (1985).

TOPP, E., SCHEUNERT, I., ALTAR, A., and KORTE, F. 1986. Factors affecting uptake of ^{14}C-labeled organic chemicals by plants from soil. Ecotoxicol. Environ. Saf. 11:219-228.

TRAVIS, C.C. and ARMS, A.D. 1988. Bioconcentration of organics in beef, milk, and vegetation. Environ. Sci. Technol. 22(3):271-274.

U.S. DEPARTMENT OF AGRICULTURE (USDA). 1982. Foods Commonly Eaten by Individuals: Amount Per Day and Per Eating Occasion. Pao, E.M., Fleming, K.H., Guenther, P.M., and Mickle, S.J. Home Economics Research Report. Number 44.

WATT and MERRILL. 1963. Composition of Foods. Agricultural Handbook. No. 8.

APPENDIX T

COMPARISON OF ANALYTICAL RESULTS FROM VERTICAL AND ANGULAR SUMP SOIL BORINGS

COMPARISON OF ANALYTICAL RESULTS FROM VERTICAL AND ANGULAR SUMP SOIL BORINGS

Analyte	Sump A-44		Sump B-10	
	Angular (10.5-14 ft.)	Vertical (10-12 ft.)	Angular (14-17.5 ft.)	Vertical (10-12 ft.)
Cd	< 3.05	< 3.05	< 3.05	< 3.05
Cr	< 12.7	< 12.7	< 12.7	< 12.7
Hg	< 0.05	< 0.05	< 0.05	< 0.05
Pb	10.9	4.39	1.4	3.85
1,3,5-TNB	< 0.488	< 0.488	< 0.488	< 0.488
1,3-DNB	< 0.496	< 0.496	< 0.496	< 0.496
2,4,6-TNT	< 0.456	< 0.456	< 0.456	< 0.456
2,4-DNT	< 0.424	< 0.424	< 0.424	< 0.424
2,6-DNT	< 0.524	< 0.524	< 0.524	< 0.524
HMX	< 0.666	< 0.666	< 0.666	< 0.666
NB	< 2.41	< 2.41	< 2.41	< 2.41
RDX	< 0.587	< 0.587	< 0.587	< 0.587
Tetryl	< 0.731	< 0.731	< 0.731	< 0.731
	Sump SC-42E		Sump SZ-4		
	Angular (7-10.5 ft.)	Vertical (10-12 ft.)	Angular (7-10.6 ft.)	Vertical (5-7 ft.)	Vertical (10-12 ft.)
Cd	< 3.05	< 3.05	< 3.05	< 3.05	< 3.05
Cr	< 12.7	< 12.7	< 12.7	< 12.7	< 12.7
Hg	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05
Pb	7.5	8.19	8.26	8.59	5.9
1,3,5-TNB	< 0.488	< 0.488	< 0.488	< 0.488	< 0.488
1,3-DNB	< 0.496	< 0.496	< 0.496	< 0.496	< 0.496
2,4,6-TNT	< 0.456	< 0.456	< 0.456	< 0.456	< 0.456
2,4-DNT	< 0.424	< 0.424	< 0.424	< 0.424	< 0.424
2,6-DNT	< 0.524	< 0.524	< 0.524	< 0.524	< 0.524
HMX	< 0.666	< 0.666	< 0.666	< 0.666	< 0.666
NB	< 2.41	< 2.41	< 2.41	< 2.41	< 2.41
RDX	< 0.587	0.669	< 0.587	0.665	0.435
Tetryl	< 0.731	< 0.731	< 0.731	< 0.731	< 0.731